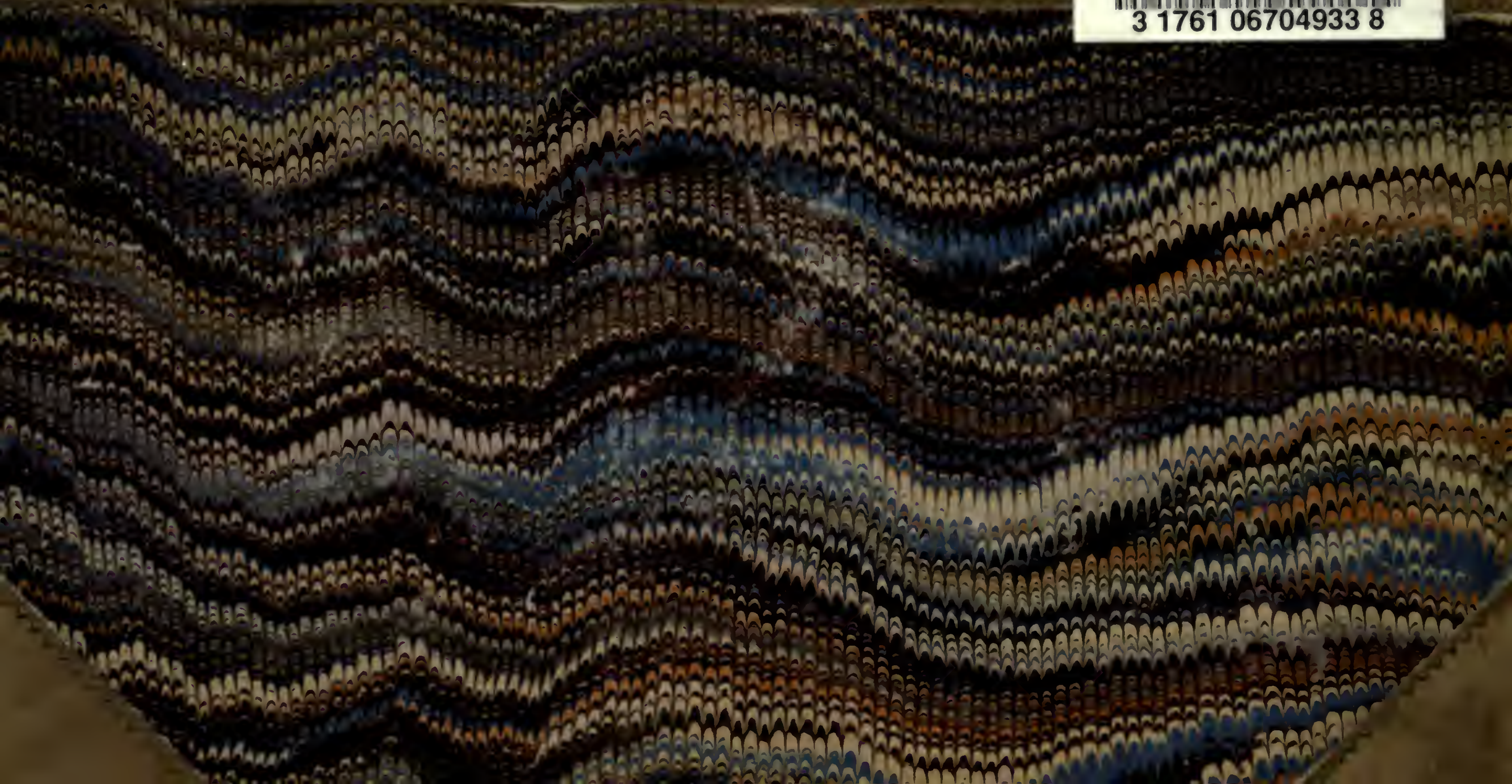

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THE DIAGNOSIS OF DISEASES

OF THE

SPINAL CORD

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THE
DIAGNOSIS OF DISEASES
OF THE
SPINAL CORD

AN ADDRESS DELIVERED TO THE MEDICAL SOCIETY
OF WOLVERHAMPTON, OCTOBER 9TH, 1879

BY
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(WITH ADDITIONS AND ILLUSTRATIONS)

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PREFACE.

THE following pages contain an address, delivered in October, 1879, to the members of the Medical Society of Wolverhampton, at whose request it is now published in a separate form. It appeared, as delivered, in the "Medical Times and Gazette" for November and December. In revising it, numerous additions have been made, in order to render the outline of the subject more complete. These additions have increased the length of the lecture, but I have thought it better to leave its form unaltered, beyond a division into sections for more convenient reference. Some illustrations have been added, which may assist the reader who is not familiar with the normal and pathological anatomy of the spinal cord. For one of these—Fig. 3, an illustration which I supplied to the eighth edition of Quain's "Anatomy"—I am indebted to the courtesy of Messrs. Longmans.

QUEEN ANNE STREET.
April, 1880.

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THE

DIAGNOSIS OF DISEASES OF THE SPINAL CORD.

Mr. President and Gentlemen,—In complying with the request with which you have honoured me, to inaugurate, with an address, another session of work of your Society, I have thought it better to select a subject of practical importance rather than of merely theoretical interest. I therefore propose to ask your attention, for a short time this evening, to the subject of the Symptoms and Diagnosis of Diseases of the Spinal Cord, having reference especially to those points in which modern investigation has added to the knowledge which is current in the profession. In order to make a description of these points useful, it is necessary to include them in a general outline of the subject, in which they may take their proper place. In such an outline it will be necessary, for the sake of clearness, to describe briefly some facts which are probably familiar. Others, which must be mentioned, may seem recondite and tedious. For both of these, therefore, I must beg your indulgence.

I trust it will not be felt that I am asking your attention to an unpractical subject. A tendency is sometimes observable, among many members of the profession, to undervalue diagnosis. Our business is to cure disease, so far as we are able, and a fear has been expressed lest our study of exactness in diagnosis should be at the expense of precision in treatment. "It matters little," it has been said, "whether your diagnosis of a diseased condition is minutely exact, if you are able to cure it." This is true; but a very superficial study of practical medicine will show that much diagnosis, which is of no direct avail for treatment, is essential for the diagnosis which enables us to treat successfully. Of all organs there are some diseases for which we can do little, there are others for which we can do much; but unless we are able accurately to distinguish the diseases of each class, we shall be unable to apply our skill where it will be effective. Moreover, there are examples of the same form of disease, in some of which the diagnosis is easy, in others most difficult. The diagnostic knowledge which is superfluous in one case is essential in another. There is, perhaps, no class of diseases to which these statements apply more truly than those which are to occupy our attention to-night.

There is another reason why a general survey of the elements of the diagnosis of diseases of the spinal cord may be useful. In systematic treatises, types of disease are described. But the mutual relations of all parts of the nervous system are very complex, and its morbid states are equally complex. Typical cases are rare, and the untypical cases are often puzzling, and can only be understood by a clear conception of the general principles of diagnosis.

The first question in the diagnosis of diseases of the spinal cord is whether the symptoms are due to organic disease or to merely functional derangement. But although this is the first question in any case, the answer to the question depends on the presence or absence of any of the signs of organic disease, and this point in diagnosis cannot be considered until we have discussed the character and significance of those signs. I would, however, impress upon you, at the outset, as a rule of cardinal importance, that the presence of a cause of functional derangement is not, in itself, sufficient ground for diagnosis. All signs of organic disease must be searched for and excluded, before the presence of the causes of functional disease is admitted as evidence. It is clear that, if there are any signs of organic disease, the existence of the causes of functional disease is of no significance whatever. Hence the importance of knowing accurately all the signs of organic disease, even the most minute and seemingly superfluous. The causes of functional derangement frequently co-exist with organic disease. Hysterical symptoms, for instance, are often present in the subjects of organic disease in all parts of the nervous system. There are two causes for this. Many organic diseases are the result of an inherited neuropathic disposition, which may also cause hysteria. Further, the damage from organic disease often affects very widely the nutrition of the nervous system, by "action at a distance"; and the degraded nutrition and function may, and often do, lead to the manifestations of hysteria. Striking symptoms of hysteria are frequently present, for instance, in cases of tumour of the brain. Hence the existence of symptoms of hysteria constitutes alone small evidence that a given disease is merely functional. It may seem superfluous to dwell upon so obvious a point, but I have seen disease again and again (and so, I doubt not, have many of you) set down as hysterical, when the plainest signs of organic disease were to be found, if looked for; and this merely because the patient, who presented certain symptoms, presented also evidence of hysteria. The same thing is true of other causes of functional derangement, and it is true also of the simulation of disease. Circumstances suggestive of malingering should be allowed no weight until the signs of organic disease are proved to be absent. Not rarely the neglect of this obvious rule has led to cruel injustice. When we think that symptoms are simulated, we should always look on our diagnosis, as well as on the patient, with suspicion, and be very sure that it is accurate, before we act upon it.

If there is evidence of the existence of organic disease, we have to ascertain further its seat and nature—to make, that is, the anatomical and pathological diagnosis. It is of importance to keep these two points distinct in our minds. Their confusion is, in all diseases of the nervous system, a fertile source of error in diagnosis. It is true that certain parts of the nervous system are frequently the seat of certain morbid processes; but to infer at once, as is often done, that because this or that region is diseased, the morbid process is of this or that character, is to make a pathological diagnosis from anatomical facts; and the diagnosis will, not rarely, be erroneous. It is true we have sometimes to use this mode of reasoning: in the absence of other evidence, or as corroborating other evidence, it is legitimate and useful, but only thus to be used, and always with a full recognition of its character and uncertainty. For instance, two patients, as I have more than once seen, will present identical symptoms of inco-ordination of movement of the legs—locomotor ataxy. This indicates disease of a certain region of the spinal cord. In the majority of cases the disease in this region is of a certain character; but in some, the symptoms being the same, the nature of the disease is quite different; and to infer the character of the morbid process in the latter case from the symptoms present, would lead us, not only to a wrong diagnosis, but to an erroneous prognosis and unwise treatment.

It is to be remembered, then, that we can only infer from the symptoms present in a case at a given time—the *seat* of the disease. To learn its *nature* we have to study the way in which the symptoms came on, and any associated conditions which may be present.

I have put this rule thus absolutely because it is one of great importance, often overlooked. There are, however, certain exceptions to it, especially the facts that pain, spasm, and sloughing of the skin are sometimes (not always) signs of an *irritative* lesion. Even here, however, the exception is rather apparent than real; for it is the acuteness of these symptoms, rather than their mere occurrence, which is of *pathological* import.

We will consider separately, then, the elements of the anatomical diagnosis, the signs which indicate the seat of the disease—"localization," as it is the custom to term it—and afterwards glance at the elements of the pathological diagnosis; that is, the symptoms which indicate the nature of the morbid process.

But we can only learn the significance of symptoms by ascertaining their nature and origin—what they are and why they are. Hence our study of diagnosis must consist to a large extent in what may be termed symptomatic pathology. The symptoms of disease are alterations of healthy function, and much of our symptomatic pathology must be, therefore, literally, perverted physiology. We must, accordingly, in the first place, have a clear conception of such points in the structure and normal function of the spinal cord, as may enable us to understand the origin of the symptoms of its diseases.

I.—MEDICAL ANATOMY OF THE SPINAL CORD.

The position of the cord, and of the origins of the nerves, in relation to the bony canal in which it lies, is the first important point which we have to consider. You will remember that the cord does not, in the adult, extend throughout the entire length of the spinal canal. It ends opposite the 1st lumbar vertebra, or opposite the interval between the 1st and 2nd lumbar vertebræ. Hence the various pairs of nerves (except the highest) do not arise from the cord opposite the vertebræ at which they leave the canal, and after which they are named, but at a higher level. The difference between the level of origin and of exit, slight in the cervical region, increases as we descend the cord, until, as you know, in the cauda equina, the lowest nerves have a very long course from the end of the cord to their foramina. It is important to know what nerves correspond in their origin with a given part of the vertebral column, because the cord often suffers secondarily to disease or injury of the bones. The relation is rendered more complex by the fact that the

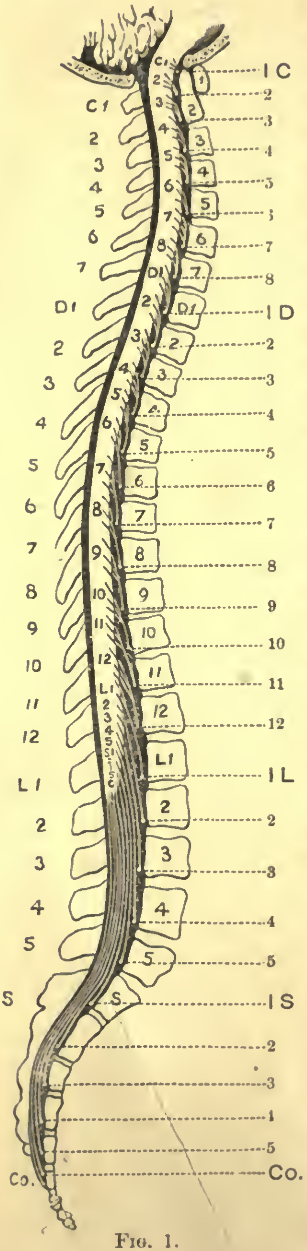

FIG. 1.

vertebral spines, which alone we can feel, and which constitute, therefore, our localizing guides, do not correspond in all parts to their vertebræ. Since these points are important in diagnosis, and are not adequately described in any English work, I have prepared a diagram (Fig. 1) showing the average relations of the spines to the bodies of the vertebræ, and of both to the origins of the spinal nerves. The tips of the cervical spines correspond nearly to the lower borders of the corresponding vertebræ. Each of the upper three dorsal spines corresponds nearly to the upper border of the body of the vertebra below. From the 4th to the 8th dorsal, each spine corresponds to the middle of the body of the vertebra below. The 9th, 10th, and 11th spines slope less, and their tips again correspond to the upper borders of the next vertebra, while the rest of the spines are opposite the bodies of their own vertebræ.

What is the relation of the spines to the nerve origins?[*] The first three cervical spines

[*] The only recorded observations on this point are those of Nuhn and Jadelot. The facts stated in the text are partly the results of a fresh examination of the relations, kindly made for me by Mr. V. Horsley, Demonstrator of Anatomy in University College.

are opposite the origins of the 3rd, 4th, and 5th cervical nerves. The 6th and 7th pairs arise opposite the intervals between the 4th and 5th, and the 5th and 6th, cervical spines respectively. The 6th cervical spine corresponds to the origin of the 8th cervical nerve, and the 7th cervical spine to the 1st dorsal nerve. The first four dorsal spines vary. The 1st spine corresponds to the interval between the 2nd and 3rd pairs, or to the origin of the 3rd pair. The 2nd spine is between the 3rd and the 4th pairs, or opposite the 4th pair. The 3rd spine is opposite the 5th, or the interval between the 5th and 6th pairs. The 4th spine is opposite the lower part of the origin of the 6th pair, or even below it. The 5th spine always corresponds to the origin of the 7th pair; the 6th spine to the 8th pair; the 7th to the 9th pair; the 8th to the upper part of the 10th pair; the 9th to the 11th pair, and the 10th to the 12th pair. The 1st lumbar nerve arises opposite the 11th dorsal spine; the 2nd lumbar opposite the interval between the 11th and 12th spines; the 3rd and 4th opposite the 12th spine; the 5th dorsal and 1st sacral opposite the interval between the 12th dorsal and 1st lumbar spines, while the remaining sacral nerves arise nearly opposite the 1st lumbar spine.

I need not describe in detail the relations of the origins of the nerves to the bodies of the vertebræ, since they may be inferred from the facts I have given, or ascertained by a reference to the diagram.

Thus the cervical enlargement of the cord, which ends at the origin of the 1st dorsal nerves, corresponds nearly to the bodies and spines of the cervical vertebræ, while the lumbar enlargement, which commences at the 12th dorsal nerves, corresponds to the bodies of the 11th and 12th dorsal and 1st lumbar vertebræ, and to the lower three dorsal and 1st lumbar spines.

We may next consider, briefly, the general structure of the cord, seen in a transverse section, such as is represented in the accompanying diagram (Fig. 2). It is divided into two halves by the anterior and posterior fissures, *af.* and *pf.* The latter is rather a septum than a fissure. The position of each is marked by a depression in the surface. In addition there are two other depressions, one where the posterior nerve-roots enter (*pr.*); another (at *s.*) about midway between this and the posterior fissure. The two "fissures" do not meet, being separated by the commissure which connects the two halves. The grey matter is, in each half of the cord, surrounded by the white substance, and is divided into two portions, or "cornua." The anterior cornu (*ac.*) varies much in size and shape in different parts of the cord, being much larger in the cervical and lumbar enlargements than in the dorsal region. It does not come to the surface, the anterior nerve-roots (*ar.*) passing irregularly through the anterior column. The posterior cornu (*pc.*, Fig. 2) is much smaller, and comes almost up to the surface at the depression (*pr.*) where the posterior nerve-roots enter. It is much larger in the lumbar enlargement than in the cervical and dorsal regions. The white substance is composed of nerve-fibres running vertically; and since these end at different levels, the white substance lessens in amount from above down. The relative amount of the grey and white substance, and the differences in size and shape of the grey cornua in various regions of the cord, may be understood from the accompanying diagrams of sections at different parts (Fig. 3).

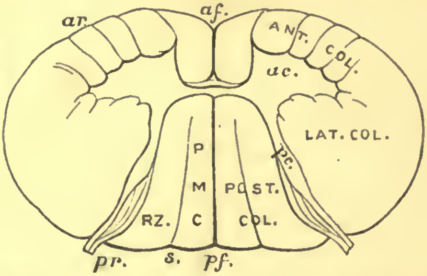

Fig. 2.—Diagram of Section of Spinal Cord in the Cervical Region. The reference letters are explained in the text.

The posterior cornua, coming to the surface, cut off the white substance between them, from the rest, and this constitutes the posterior columns. Each posterior column thus lies between the posterior median septum and the posterior cornu. The posterior roots of the nerves do not all immediately enter the grey substance, but some of them course through the outer part of the posterior column, which we may term the postero-external column, or, following Charcot, the "posterior root-zone." (RZ., Fig. 2) A septum

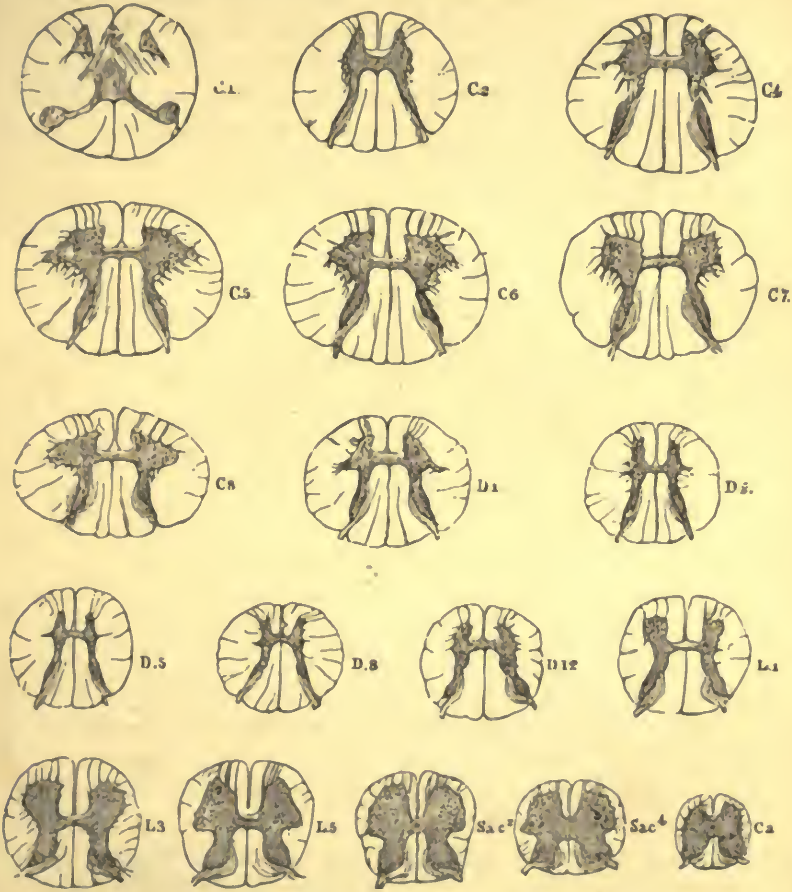

FIG. 3.—DIAGRAMS OF SECTIONS OF THE SPINAL CORD AT DIFFERENT LEVELS.

The letters and numbers indicate the spinal nerves to which the sections correspond. Each is figured twice the natural size. (From Quain's "Anatomy," 8th Edition.)

of connective tissue (*s.*, Fig. 2) separates off, from this area, that part of the posterior column which is adjacent to the posterior median fissure, and the part so marked off is termed the "posterior median column." The distinction of these two portions of the posterior column is, as we shall see, very important in pathology. But it is to be noted that the fibres of the posterior roots only pass through the outer part of the postero-external column, and that these fibres in the lumbar region pass further into the column than the cervical region (Compare c 6 and L 5 in Fig. 3).

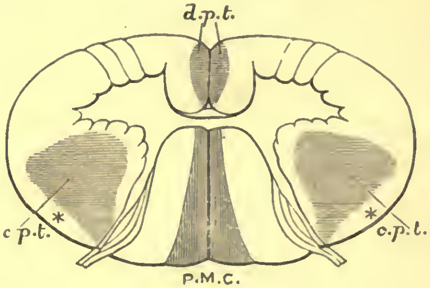

FIG. 4 —AREAS OF SECONDARY DEGENERATION.

P.M.C., postero-median columns, one on each side of the posterior median septum; *d.p.t*, direct pyramidal tracts, one on each side of the anterior median fissure; *c.p.t.*, crossed pyramidal tract in the posterior part of each lateral column, and separated from the surface of the cord by (*), the direct cerebellar tract of Flechsig. The areas of ascending degeneration are shaded vertically: those of descending degeneration transversely.

The portion of white matter which lies in front of and outside the grey, from the anterior median fissure to the posterior cornu, is structurally undivided, and is termed the antero-lateral column. It has been artificially divided into an anterior column, lying to the front and inner side of the anterior cornu, and a lateral column, lying outside the grey matter (Fig. 3). But pathology indicates a more important division than this, and the study of the development of the cord corroborates the teachings of pathology. If certain parts of the brain (concerned in voluntary motion) are destroyed, certain fibres degenerate throughout the cord, and this degeneration thus marks out for us the fibres which are in direct connection with the motor region of the brain. Two tracts are thus picked out, one in the posterior part of the lateral column, on the opposite side to the cerebral lesion; and one on the same side, in the anterior column, close to the median fissure (see Plate, Fig. 1, *a* and *b*). These are called the "pyramidal tracts," because the connection of these tracts with the brain is by means of the anterior pyramids of the medulla. They are shown on both sides, shaded transversely, in Fig. 4. Those adjacent to the anterior median fissure are the "direct pyramidal tracts"; those in the lateral column are "crossed pyramidal tracts."

The crossed pyramidal tract contains the motor fibres which have decussated in the medulla; the direct tract, those which have not decussated there. The relative size of these tracts varies in different individuals; the more fibres that have crossed in the medulla, the smaller is the direct tract, and *vice versâ*. The direct tract may even be absent, all the fibres having crossed above (Flechsig).

Regarding the crossed pyramidal tract, it may be observed that it is situated behind the level of the anterior cornu, that it does not usually extend quite up to the posterior cornu (although it may do so behind), and that it never extends up to the surface of the cord, being limited by a zone (* Fig. 4), in which there is no descending degeneration. The fibres of this zone are said (by Flechsig) to descend from the cerebellum.*

We have further evidence that the fibres in the posterior parts of the lateral columns descend from above, in the fact that, if the cord is destroyed at any level, these fibres on each side degenerate below the lesion, just as they do on one side after a cerebral lesion. Such bilateral degeneration is shown in Fig. 5, C, *c c*, the lesion causing it being indicated at A. Bilateral degeneration of these tracts is shown also on the Plate, Fig. 2, *b b*. This degeneration is currently, although not very happily, termed "sclerosis," and the degeneration of this area is designated "lateral sclerosis"—"descending lateral sclerosis," when it is the result of a lesion higher up.

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* The direct pyramidal tract is also called the column of Türck; the postero-median column is called the column of Goll, and the root-zone (postero-external column) is called the column of Burdach. I have avoided the use of these terms. This system of nomenclature is one full of inconvenience, increasing the difficulties of the student, and leading to frequent mistakes in scientific writings. There are very few observations in medicine regarding which it is not obvious that they would speedily have been made by some one other than the actual observer; that it was very much of an accident that they were made by certain individuals. Scientific nomenclature should be itself scientific, not founded upon accidents. However anxious we may be to honour individuals, we have no right to do so at the expense of the convenience of all future generations of learners.

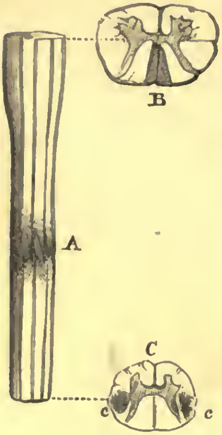

FIG. 5.

I mentioned that the fibres of the white columns end at different levels, and so the white columns become progressively smaller. The portions of the white columns which constitute the pyramidal tracts follow the same rule, and hence the descending degeneration becomes smaller in area the lower we descend in the cord, and in the lowest part of the lumbar enlargement it is very small indeed. The fibres of the direct pyramidal tracts disappear in the dorsal region, probably passing into the grey matter, and decussating. Hence the descending degeneration from the brain, in the lower dorsal and lumbar region, is confined to the opposite lateral column. Hence, too, if the cord is compressed, the descending degeneration appears in the direct pyramidal tracts only when the compression is high up.

A similar process of "secondary degeneration" furnishes corroboration of the distinctive division of the posterior column, which, as we have just seen, is suggested by anatomy. Below a point at which the cord is completely destroyed, although the lateral columns degenerate, the posterior columns present no change. Above the point destroyed, however (as at B, Fig. 5), while the lateral columns, and the root-zone of the posterior columns present no change, in the postero-median columns the nerve-fibres disappear and become replaced by fibrous tissue.* This ascending degeneration is also shown in the Plate, Fig. 3, *c*. These are the only secondary degenerations commonly described. But I have lately found, in a spinal cord of which the lower extremity was crushed, a symmetrical area of slight ascending degeneration in the anterior part of the lateral columns, in front of the pyramidal tracts (Plate, Fig. 3, *e*). Of its possible significance I will speak presently.

The grey substance is composed of nerve-cells and interlacing fibres; some of the cells in the anterior cornua are very large and with many processes, and are called the "ganglionic" or "motor" nerve-cells, because at least some of the anterior, motor, nerve-fibres are connected with them.

II.—PHYSIOLOGY OF THE SPINAL CORD IN RELATION TO THE SYMPTOMS OF ITS DISEASES.

We may now consider the chief functions of the cord, and the effects of their impairment. In the spinal functions we have to distinguish two great systems of action—that by which the cord transmits, and that by which it controls; *i.e.*, its functions as a conducting organ, and as a nerve-centre, reflex and automatic.

Motor Conduction.—The conduction of motor impulses from the brain is in the antero-lateral white columns, perhaps solely in the pyramidal tracts; it is chiefly in the side of the cord corresponding to the limbs moved, the crossing taking place for the most part in the medulla. The motor path leaves the cord by the anterior nerve-roots, but does not enter them directly, passing into the grey matter and probably through the motor nerve-cells with which the anterior roots are connected. The power of voluntary motion may be arrested by a lesion anywhere in this tract—lateral column of the cord, grey matter, and anterior nerve-roots. If the lesion is on one side of the cord, the loss of power will be on the same side, and in degree proportioned to the number of pyramidal fibres which have crossed in the medulla; and this, as we have seen, is not always the same.

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* This statement, although that which is current, is not strictly accurate. Some distance above the damage the ascending degeneration is confined to the postero-median columns; but close above the compression the degeneration extends outwards into the posterior portion of the postero-external column, not, however, to that part of it through which the posterior roots pass. Hence it is probable that the fibres which course upwards in the postero-median column enter it from the postero-external column.

Sensory Conduction.—All sensory impulses—of pain, touch, temperature—enter the cord by the posterior roots, passing, in part directly, in part through the root-zone of the posterior columns, into the posterior cornu, and quickly crossing to the other side of the cord. There is some reason to believe that the paths of these several sensory impulses up the cord are not the same. That of pain has been commonly believed to pass up the central grey matter; that of touch, and perhaps also of temperature, passes up, in the opinion of some authorities, in the posterior column. But according to late, most careful, and apparently conclusive experiments by Woroschiloff (confirmed by Ott), such sensation as can be tested in the lower animals is conducted, in the dorsal region, in the lateral columns. No facts have been hitherto recorded suggesting that this is true of man. But if sensation is conducted in part in the lateral columns, it is certainly not in that portion of them which is occupied by the pyramidal tracts, because there may be no loss when these are completely degenerated. It is probably, therefore, in front of these. This is the situation in which I have found the ascending degeneration in the case of crushed cord in which sensation was greatly impaired. (See p. 13, and Plate, Fig. 3, *e*.) This fact at present stands alone, but, taken in conjunction with the experiments on animals, it points, I think, to the probability that some sensation is conducted in this region in man; what or whence, whether from the skin or deeper structures, we do not know.

We are still too ignorant of the paths of sensation for us to infer much from the form of its affection in spinal disease. One thing, however, seems clear—the path of sensation is less determined than that of motion. A very small portion of undestroyed cord will conduct sensation, but it is then, at least in its intenser form, commonly retarded. Thus each form of sensation may be impaired by disease of the posterior roots, either outside the cord or in the root-zone through which they pass; or by disease of the conducting structures of the cord higher up; and, since the paths decussate in the cord, if the lesion is unilateral, sensation will be affected on the side of the body opposite to the lesion (motion being affected on the same side). A strong reason for believing that the paths are not the same is, that the senses of touch and pain and temperature are often impaired in different degrees. The most common change is for the sense of pain to be lost, and touch preserved (analgesia). In such a condition the slightest touch of the finger may be felt readily, but a needle may be driven into the skin and the patient experience only the sensation of a touch. In other cases the sense of touch may be lost, and only the perception of painful impressions remain (anæsthesia). In other cases both are changed proportionately. To ascertain impairment, it is necessary, therefore, to examine carefully the sensitiveness to each form of stimulation, to note how the patient feels the impression (since the sensation, when not lost, may be perverted) to note whether it is localized accurately, and to note whether it is unduly retarded. Sensations of pain and temperature are never so rapid as that of touch, and it is in these that the chief retardation takes place.

The functions of the ascending fibres of the postero-median columns are still unknown. Their degeneration does not seem to be accompanied by any impairment of sensation.

Reflex Actions.—The next important function of the cord is its action as a reflex centre. We may regard the reflex system of the cord as made up of a series of nerve-loops, each posterior, sensory, root being connected with certain anterior, motor, roots by means of the grey matter (Fig. 6). This consists partly of the large motor nerve-cells, and partly of a network of the finest nerve-filaments and minute nerve-cells. The connection of the roots, through the grey matter, is apparently by this network of interlacing and connected fibres, infinitely numerous, like the filaments of a sponge.

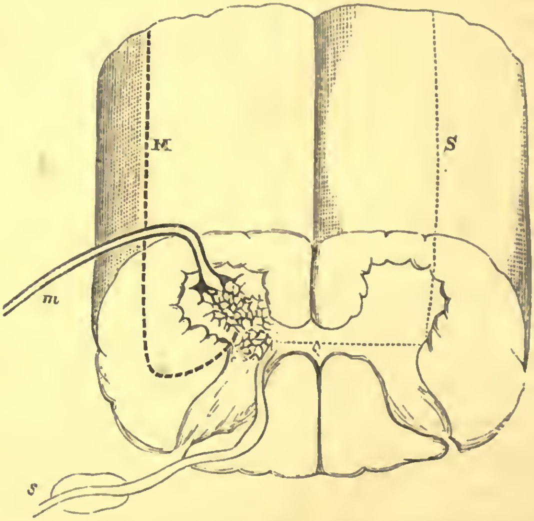

Fig. 6.—Diagram of a Reflex Loop.

M, conducting motor tract; *m*, anterior motor nerve-roots; S, conducting sensory tract; *s*, posterior sensory nerve-roots.

But in this are paths of different resistance, so that a slight stimulus may pass by the most ready path to a certain anterior root, and a stronger stimulus may diffuse itself more widely and affect many nerve-roots. For instance, a gentle touch on the sole may cause only a movement of the toes; a stronger touch, a start of the whole leg. A similar wide diffusion may occur in pathological states of the cord. These reflex loops are also connected with the conducting tracts to and from the brain. A motor impulse, passing down the cord in the white column (M), leaves the cord by the anterior roots (*m*), which are part of the reflex loop, and probably enters the anterior roots by the motor nerve-cells, which may be regarded as part of the reflex centre. So, too, the sensory impulse enters the cord by the posterior nerve-roots (*s*), which are also part of the reflex loop, and then, leaving this loop, ascends up the opposite side of the cord to the brain. Thus the same peripheral impression excites a conscious sensation and a reflex action; and, on the other hand, we can, if we wish, execute voluntarily a movement of the leg quite the same as that of the reflex act. Moreover, we can exercise some voluntary control over the reflex action, and prevent the start of the leg.

The value of the reflex actions in diagnosis is, that their persistence is proof that there is no considerable disease in the reflex loops by which they are produced. Hence they yield us very important diagnostic information. Their absence or their excessive degree is, in some circumstances, equally important. It will be well, therefore, to study them in more detail.

It is necessary to distinguish two forms of reflex action—the superficial and the deep. The superficial is that excited by stimulation of the skin, by a touch, scratch, prick, etc. On gentle stimulation, contraction occurs in the muscles at or near the spot. A series of such reflex actions can often be obtained in the normal spinal cord, from the lowest extremity of the cord to the lower part of the cervical enlargement. In some cases they are of considerable diagnostic importance. Beginning below, we have the well-known reflex from the sole (plantar reflex) which depends on the lower part of the lumbar enlargement, when the movement which results is confined to the foot-muscles. (See Table, p. 52.) Next, irritation of the skin of the buttock, in some individuals, excites a contraction of the glutei—the gluteal reflex, we may call it—depending, I believe, on the cord at the level of the 4th or 5th lumbar nerves. Next, there is the well-known cremaster reflex, by which the testicle is drawn up when the skin on the inner side of the thigh is stimulated. This arises at the level of the 1st and 2nd lumbar pairs. It may often be excited by stimulation of any part of the front and inner side of the thigh.* Next, there is the abdominal reflex—a contraction in the abdominal muscles, when the skin is stroked on the side of the abdomen, from the edge of the ribs downwards. This is produced in the cord from the 8th to the 12th dorsal nerves. Next, a stimulation on the side of the chest, in the 6th, 5th, and sometimes in the 4th intercostal spaces, causes a dimpling of the epigastrium on the side stimulated. I think that it depends on a contraction in the highest fibres of the rectus abdominis; it is singularly uniform in its occurrence. We may term it the epigastric reflex; it depends on the spinal cord from the 4th to the 6th or 7th pairs of dorsal nerves. There is no higher reflex on the front of the trunk. If we turn to the back, we shall find that in some patients, from the angle of the scapula to the iliac crest, stimulation of the skin along the edge of the erectors of the spine excites a local contraction in these muscles. These dorsal and lumbar reflexes, as they may be termed, are only of corroborative value, as they are less active than the more convenient abdominal and epigastric reflexes, which are produced in the same region of the cord. Irritation of the skin in the interscapular region gives us, however, the highest reflex available—a contraction in some of the scapular muscles, when slight, chiefly marked at the posterior axillary fold (teres); when more considerable, involving almost all the muscles attached to the scapula—trapezius, teres, serratus—and even moving the bone a little outwards. We may term it, therefore, the scapular reflex, and it is produced in the cord at the level of the upper two or three dorsal and lower two or three cervical nerves.

Thus in these reflexes—plantar, gluteal, cremasteric, abdominal, epigastric, and scapular—we have the means of ascertaining something of the condition of almost every inch of the spinal cord from the cervical enlargement downwards.

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* This reflex has been carefully studied by Jastrowitz, and lately by Weir Mitchell ("Am. Journal Med. Sci." Oct. 1879).

The presence of the reflexes is proof that the path through the cord is not seriously uninterrupted, but we cannot *simply* infer from their absence that this path is impaired. The reflex excitability of the cord varies much in different individuals, is always greatest in early life, and is often lessened in the old. Some of these reflexes are thus absent, apart from disease, especially the gluteal and lumbar reflexes, and sometimes the cremaster reflex; the abdominal reflex is also lessened by laxity or distension of the abdominal parietes. It is a remarkable fact, also, that disease of one cerebral hemisphere lessens or abolishes these superficial reflexes on the opposite (paralysed) side of the body—a fact which has been very carefully studied, as regards the abdominal reflex, by Rosenbach, and as regards the cremasteric reflex, by Jastrowitz. It is an effect very difficult to explain, because these reflexes are increased if the disease, which lessens voluntary power, is not situated in the brain, but is high up in the cord. There is, I think, only one possible explanation. In the frog the superficial reflexes are controlled by a centre, situated in the optic lobes, and are lessened, or at least retarded, if this is stimulated. It is probable, as just observed, that there is also in the higher animals a centre which has the power of controlling these reflex actions, and that, in man, this is situated, not in the corpora quadrigemina, but in the optic thalami. If we assume that this controlling centre is itself under the influence of the highest motor centres—not an improbable assumption—all the phenomena are intelligible. The motor centres normally restrain the controlling centre: if the motor centres (or path from them to the controlling centre) are damaged, this centre is unrestrained, and inhibits the superficial reflexes on the paralysed side. But disease in the cord interrupts, not only the voluntary path, but also that by which the controlling centre influences the superficial reflexes, and so these are intensified in the paralysed parts. The effect of cerebral disease does not interfere materially with the use of these reflexes as indications of spinal disease, and it affords us an important additional indication of the existence of an organic disease of the brain. I will presently give you some instances of the utility of these reflexes in spinal diagnosis.

We may now pass to the consideration of the deep reflexes. These are also of great importance, physiological and pathological. The afferent impressions which excite them originate from structures deeper than the skin—from tendon, from muscle, and perhaps also from joints. The group includes those which have been called "tendon reflexes," a term objectionable as a general designation, because, as we shall see, the relation to tendon of some which have been called by the name is exceedingly doubtful; and of all it is true that they are not exclusively related to tendon.*

The best known of these reflexes is that which has been termed the "patellar tendon reflex," or "knee phenomenon," or "knee reflex." The latter is, I think, the preferable term. If, when the knee is flexed so that the leg is free to move, the quadriceps femoris being gently extended, the patellar tendon is struck, the quadriceps contracts and jerks the leg forwards. The most convenient position is with the knee to be tested flexed nearly, but not quite, at a right angle. It should not hang vertically, or the swing caused by the blow may be mistaken for the reflex movement. It must be free to move, or a slight reflex jerk will not be perceived. The posture commonly employed is with the leg to be tested across the other, the knee of the supporting leg being at a right angle (Fig. 7). But if the leg to be tested is stout, its tension in this position may be too great to permit of any movement. In such case the best posture is for the observer to place his arm beneath the patient's thigh, just above the knee, and rest his hand on the patient's other knee (Fig. 8). Not long ago I saw a rather stout man, well known to many members of our profession, who was uneasy because a physiological friend had been unable to produce this reflex upon him. His legs were so stout that, in the posture commonly employed, no movement occurred when the patellar tendon was struck. But when the thigh rested on the observer's arm, in the way I have described, the tap on the tendon caused a ready reflex, much to the satisfaction of the individual examined, whose anticipations of impending locomotor ataxy were thus removed. Children may sit on the edge of a chair, but if so, and the legs are vertical, the swing and the reflex must be carefully distinguished. The side of the extended hand is a convenient instrument for giving the blow (Fig. 7). Now and then, when very slight, a percussion hammer (Fig. 8) elicits it more readily (as Erb, abroad, and Buzzard, in this country, have pointed out). I have never, however, succeeded in obtaining it with the hammer, and not with the hand, except in cases in which the space between the patella and tibia was too small to permit of a suitable blow with the hand. It may commonly be obtained as

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* For a knowledge of these phenomena we are largely indebted to two distinguished German physicians, Erb and Westphal. Attention was first called to them in this country by Dr. Grainger Stewart, subsequently by Dr. Buzzard. The evidence on which the following view of their nature is based will be found in a paper by the author in the last volume of the "Medico-Chirurgical Transactions" (1879).

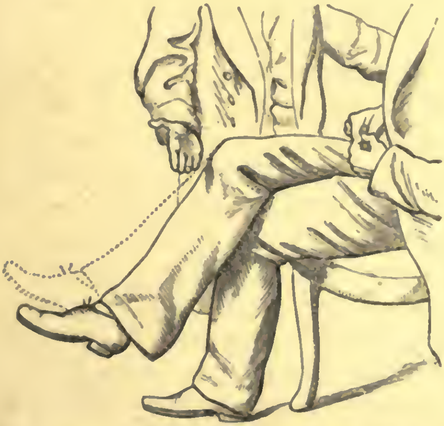

Fig. 7.—Ordinary mode of Obtaining the Knee Reflex.

The dotted line indicates the movement which follows the blow on the patellar tendon.

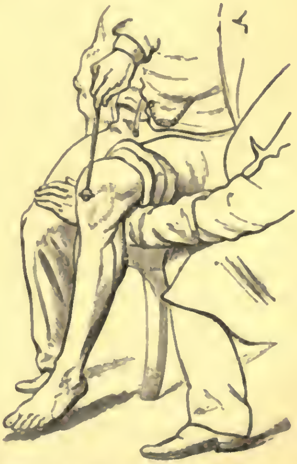

Fig. 8.—Knee Reflex.

Method of obtaining it with a percussion hammer when it is not readily produced in the ordinary way.

readily through one or two garments as upon the skin. If its existence is doubtful, however, the skin should be bared. In many cases the reflex may be obtained by a downward blow upon the patella, by a blow on the quadriceps tendon above the patella, or by a blow on the substance of the muscle, almost as readily and strongly as by a blow on the patellar tendon. In cases in which it has been in great pathological excess, I have even excited it readily by a blow on the tibia.* When the patellar tendon is tapped, the afferent impulse probably originates from the tendon, but these facts show that in some cases it may originate away from the tendon. Hence I think that the term "knee reflex" is a better designation than "patellar tendon "reflex."

It is, without doubt, a true reflex action, depending on the integrity of the reflex loop at the level of the second and third lumbar nerves. It is very readily impaired by disease of any part of this loop—(1) of the posterior nerve-roots, outside the cord, or in the posterior column (and hence it is commonly, though not invariably, lost in locomotor ataxy). It is impaired by disease (2) of the grey matter, or (3) of the anterior roots, or (4) of the mixed nerve-trunk. It is occasionally, as I have seen, absent in normal conditions; perhaps in about one individual in a hundred (Berger). It is excessive in some forms of cord disease, especially in those in which the descending degeneration occurs in the lateral columns. Thus it is excessive in hemiplegia on the weakened side, and is therefore changed in the opposite way to the superficial reflexes, which we have seen are lessened in hemiplegia. It is excessive on both sides when there is disease higher up the cord, causing descending degeneration.

The next important phenomenon belonging to this group occurs at the ankle-joint, and its importance and significance are very great. It has been termed the "Achilles tendon reflex," but it is, I believe, in the highest degree doubtful whether, as ordinarily observed, it has anything to do with the Achilles tendon. It is best studied in cases in which it is in excess—the same class of cases which present excess of the knee reflex. In many of these, if the calf muscles, which extend the ankle-joint, are suddenly put on the stretch by pressing the hand against the sole of the foot, a quick contraction occurs, instantly ceasing, but, if the pressure is kept up, instantly renewed, and recurring, as long as the tension is maintained, as a clonic series of spasmodic contractions—the "ankle clonus." It can often be obtained best when the knee is not completely extended. The movement is very uniform, from five to seven contractions occurring per second. By attaching a writing point to the foot, and making it trace a line on a revolving cylinder of blackened paper, I have obtained such tracings as I now

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* The same fact has been observed by Dr. Byrom Bramwell (*Medical Times and Gazette*, vol. ii. 1879).

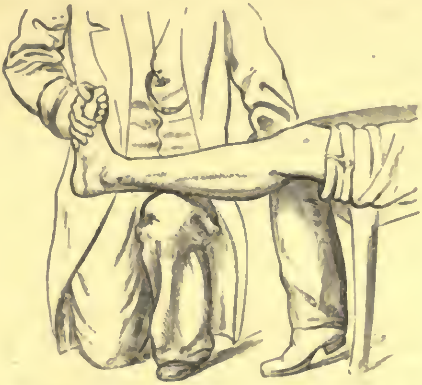

FIG. 9.—METHOD OF ELICITING THE ANKLE CLONUS BY PASSIVE FLEXION OF THE FOOT.

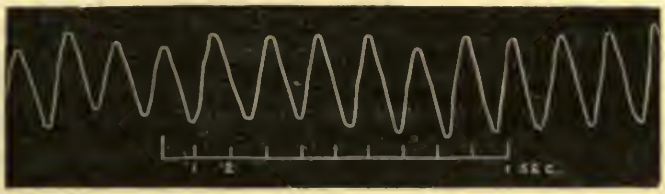

FIG 10.—TRACING OF THE ANKLE CLONUS IN PARAPLEGIA. (The tracing reads from right to left.)

show you (Fig. 10), which are almost as regular as the tracing obtained from a tuning-fork. How these contractions arise is a matter of some dispute. They are evidently excited by the sudden tension, the effect of the continued tension exciting, on each relaxation, a fresh contraction. If the tension be applied very gently, no contraction occurs, but then, if the Achilles tendon is tapped, the muscle instantly contracts. Hence it was assumed by Erb that the sudden tension excites a contraction by stimulating the nerves of the tendon, and that each contraction is reflex. But, during the passive tension, a tap on the calf muscle itself will cause a similar contraction, and so also, I have found, will a tap (Fig. 11) on the muscles in front of the leg (tibialis anticus), and it is not conceivable that the muscle-tap and the front-tap stimulate the tendon. Moreover, I have found that the time which elapses between these three taps and the resulting contraction (·03 or ·04 sec.) is too short for the briefest reflex process (according to received physiological facts). Hence it seems probable that every contraction is produced by the local stimulation of the fibres of the muscle, the passive flexion exciting them by the sudden tension, the tap on the Achilles tendon also increasing their tension or sending a vibration through them, the tap on the muscle and on the front of the leg sending a

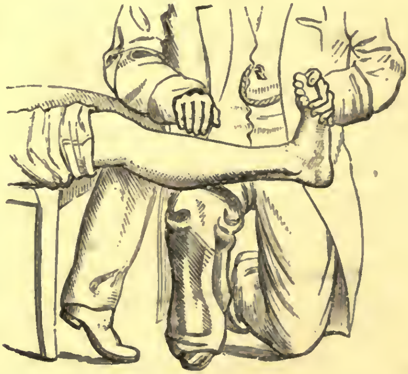

FIG. 11.—METHOD OF OBTAINING THE FRONT TAP CONTRACTION.

slight vibration through the muscle (which may be felt by the hand applied to the calf), which excites a contraction in the state of extreme irritability. This extreme irritability, however, has also to be explained. It is clearly the result of the passive tension placed upon the muscle, and it is, perhaps, a true reflex phenomenon, the afferent impulse being the tension on the muscular fibres. It is thus assumed that the passive tension may produce two effects:—(1.) To cause, by reflex action, an excessive irritability to local stimuli—a state of incipient contraction in which an actual contraction is developed with extreme readiness. If the tension is gradual and gentle, the effect may be confined to this. (2.) If sudden, the tension excites not only the reflex irritability, but also a local contraction. When this contraction is over, the continued tension on the relaxing fibres developes another contraction, and so on in the recurring clonus.* Additional evidence that the afferent impulse originates in the muscle, and not, as has been supposed, in the tendon, is furnished by the fact (which you may easily verify on yourselves) that the passive flexion of the foot causes a sensation of distinct pain in the muscle, and no sensation in the tendon—the pain proving that an afferent impulse does originate in the muscle under the conditions. The same conclusion is indicated by the fact that, while a lateral tap on the tendon will cause the contraction, if the other edge of the tendon is so supported that the tendon cannot move under the tap, and so cannot affect the muscle, no contraction occurs. If it should be ultimately proved that the short interval which I have found to intervene between the tap and the contraction is sufficient for such a reflex process, each contraction may be reflex, but it will remain true that the impulse exciting it is from the muscle, and has little or nothing to do with the tendon, and that the term "tendon reflex" is an inaccurate and misleading term. It is essentially a muscular reflex phenomenon, not a tendon reflex.

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* In this connection, it is interesting to note that physiologists have ascertained that a moderate resistance, or "load," increases the contraction which results on a given stimulation through the nerve. (See Foster's Physiology," 3rd Ed, p. 81.)

I would call especial attention to the "front-tap contraction," the contraction which occurs when (during passive flexion of the ankle) the muscles in the front of the leg are tapped (Fig. 11). It may be obtained when the clonic spasm cannot be set up, and constitutes a very delicate and convenient test of morbid irritability.

A clonus quite similar to that just described can be sometimes obtained in the peronei (a lateral ankle clonus), and also in the plantar muscles of the great toe—in each case by passive tension. All have nearly the same time—about six per second.

These are not entirely pathological phenomena. In health the front-tap contraction may sometimes, although very rarely, be obtained. The tendon-tap contraction may sometimes be obtained. The ankle clonus *can never be obtained in health by sudden passive tension.* Thus produced it is absolutely pathological, and of the highest importance as certainly indicative of a structural change in the spinal cord. Its diagnostic importance can hardly be over-estimated. In many cases in which it occurs, the nutrition and sensibility in the legs are unimpaired, and the weakness in the legs is likely to be regarded as "functional," or, in a woman, as "hysterical." I have seen many such cases, thought to be hysterical, in which it needed but a touch on the sole of the foot to excite the ankle clonus—an absolute proof of the existence of organic disease.*

But although, thus produced, the phenomena are pathological, we may get evidence of the same kind of reflex action, in health, in another way. If a rhythmical contraction can be set up voluntarily, and gentle tension in the gastrocnemius maintained, as by sitting on the edge of a chair with the ball of the foot resting on the ground, the contractions will go on involuntarily—a normal ankle clonus, which has precisely the same time (about six per second) as the morbid clonus (Fig. 12). It is evidently the same phenomenon, the difference being that it cannot in health be excited by passive tension. For *this* to be effective a morbid reflex irritability is needful, such as only exists in disease. In ataxies, in which the knee reflex is lost, this normal ankle clonus cannot be obtained; *i.e.*, the voluntary movement does not, as in health, continue involuntarily. In morbid states, in which the deep reflexes are in excess, the posture I have just described excites the clonus very readily; and the jerking legs of paraplegics as they sit must be familiar to you. In attempting to walk, also, the tension on the calf-muscles has the same effect, and the patient may be jerked violently by the spasm.

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* The only apparent exception to the rule is the fact that it may rarely be obtained in cases of chronic rheumatic joint-affection. But in all such cases which I have seen there has been other evidence that the joint-affection has been accompanied by secondary changes in the spinal cord.

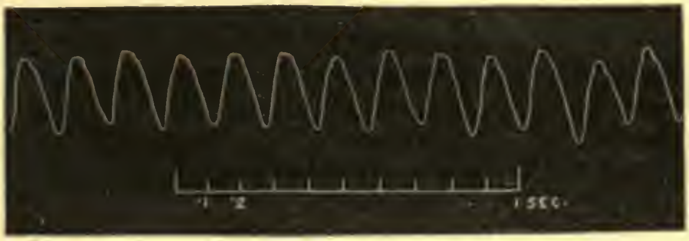

FIG. 12.—NORMAL ANKLE CLONUS.
(The tracing reads from right to left.)

Thus I think that these phenomena depend on the fact that passive tension in the muscle developes, by a reflex mechanism, a state of local irritability, it may be of slight tonic contraction in it, in which a single contraction may be locally excited with great readiness. It is probable that this is a part of a general system of muscular reflex relation between tension and contraction, of the highest importance in the associated action of muscles, and that the reason why, in certain muscles, as those of the calf, these phenomena are more readily observed, is because in these, in the act of walking, at every step contraction succeeds tension, and so the reflex relation between the two has attained a higher degree of development. I have never seen the ankle clonus in a child who had never walked.*

The so-called "tendon reflexes" which occur in the arm are not of importance in the diagnosis of disease of the spinal cord. Before leaving these spinal reflex phenomena, I may, parenthetically, illustrate to you, by an example, the manner in which they are changed in cerebral disease, and the occasional diagnostic importance of their alteration. As I told you, the superficial reflexes, especially the trunk reflexes, are lessened, and the deep reflexes are increased, when there is any degree of descending degeneration in the cord. Under these circumstances the ankle clonus and front-tap contraction may be obtained. Not long ago, in investigating these reflexes, I examined a man who was thought to be suffering from idiopathic epilepsy. No weakness was complained of. The epigastric and cremasteric reflexes were, however, absent on the right side, and the abdominal reflex was slight, although all were well marked on the left. In the right leg the knee reflex was excessive, and the ankle clonus and front-tap contraction could readily be obtained, while they could not on the left. This alteration affords the strongest grounds for suspecting organic brain disease, and its discovery led to careful examination of the strength of the right limbs. Slight but distinct weakness of the right arm and leg was found, and has since increased, and optic neuritis is now developing. In this case organic disease of the brain—probably a tumour—certainly exists; but it might readily have been overlooked, and probably would have been overlooked for a time, had it not been for the indications afforded by these reflexes.†

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* Dr. Byrom Bramwell has, however, observed an ankle clonus in a child who had never walked. It remains true, I think, that the clonus is much rarer in such patients than in adults in similar pathological states.

† In this patient the right hemiplegia and the optic neuritis increased and ultimately left hemiplegia gradually supervened. After death there were found tumours in each hemisphere of the brain. That on the left side, which was the larger, the size of a pullet's egg, was situated above the lateral ventricle, beneath the upper extremity of the ascending frontal convolution, and it had extended through the lateral ventricle to the surface of the optic thalamus. It had clearly interrupted the connection of the upper "motor convolutions" with the motor tract and with the optic thalamus.

Co-ordination of Movement.—The next function of the spinal cord to be mentioned is that by which it influences the co-ordination of muscular movements. This function depends chiefly upon the posterior columns, and is lost when there is disease in this situation, as in locomotor ataxy. It is not, however, the whole of the posterior columns which are related to the function, but merely the part we have distinguished as the postero-external column or root-zone, that through which the fibres of the posterior roots pass. The most complete ataxy may result from disease limited to this situation (Plate, Fig. 5). It is not probable that movements are, strictly speaking, co-ordinated in the cord: the probable seat of this function is the basal ganglia of the brain. Disease of the posterior columns may interfere with the influence of the co-ordinating centres upon the muscles or lower centres. But we have seen that, by means of the deep muscular reflex actions, muscular contractions become associated; tension influences contraction; and thus there is a reflex grouping of muscular actions, which undoubtedly plays a very important part, not only in the actual arrangement of the contractions, but also by, so to speak, moulding the spinal centres by establishing lines of lessened resistance through them, and so facilitating the voluntary co-ordination.

There is, I believe, another mechanism by which the impairment of reflex action may impair co-ordination. For any movement there must be not only a contraction of certain muscles, but also a proportioned relaxation of their opponents. I have lately* adduced evidence to show that this relaxation is really due to a reflex action, and the same thing is suggested by the fact, long ago pointed out by Duchenne, that the late rigidity of hemiplegia may commonly be readily relaxed by faradization of the opponents of the rigid muscles. If this is so, we obtain a glimpse of a very complex series of myo-reflex actions, by which a relation is established between tension, contraction, and relaxation, which must undoubtedly play a very important part in muscular co-ordination. These are all dependent on the deep reflex processes, which in locomotor ataxy are almost always impaired (if we are to judge by the knee reflex which is commonly and early lost). Hence it is a question whether the loss of these reflexes will not alone account for the inco-ordination in posterior sclerosis, without the assumption of disease of special co-ordinating fibres, which have been supposed to run vertically in the postero-external columns.

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* "The Movements of the Eyelids"—"Med.-Chir. Trans." 1879.

But there are cases in which ataxy exists without loss of the knee reflex, and with the ankle clonus, *i.e.*, with some, at least, of the deep reflexes in excess. These cases, of which I have recorded several,* are very difficult to harmonize with the theory that ataxy depends solely on the impairment of the deep reflexes. But it may be remarked that the inco-ordination is in these cases never quite like that in typical ataxy—there is more unsteadiness than inco-ordination. It may be that the deep reflexes are impaired elsewhere than the region in which the knee reflex and ankle clonus are developed.

However, I would only insist on one point—that we know nothing of co-ordination of movements in the spinal cord of man, except as the result of the deep reflex actions; † and that if there is more in locomotor ataxy (posterior sclerosis) than this loss, it is due to an interference in some way with cerebral co-ordination. Of course, when superficial sensation is lost from the extensive damage to the posterior roots, this loss will greatly increase the ataxy by the loss of an important indication for cerebral guidance of movement. But since ataxy may exist without loss of superficial sensation, this is evidently not the *chief* element in the condition.

In the inco-ordination of movement which depends on the spinal cord, we greatly intensify the manifestation of the trouble by reducing the base of support, since the smaller this is, the more accurate is the adjustment of muscular actions needed to maintain perfect equilibrium. Hence the ataxic has a difficulty in standing with his toes and heels close together; and if his feet are uncovered, the irregular muscular contractions are obvious in the twitching of the tendons. Moreover we increase his difficulty by making him close his eyes, withdrawing, thus, the visual guidance. It has been said that this test is only effective when sensation is impaired in the legs, *i.e.*, when the sensory impressions from the legs are insufficient for cerebral guidance. This is, however, not true. It may be very marked when sensation is perfect. The maintenance of equilibrium is partly a deep reflex act, and if this function is slightly impaired, we can easily understand that it should be inadequate, unless supplemented by the visual information.

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* "Med.-Chir. Trans." 1879, p. 280.

† The springing movements observed by Woroschiloff in the hind legs of the rabbit after section of the dorsal cord, and regarded by him as "co-ordinated" in the lumbar enlargement, were probably merely a consecutive series of myo-reflex actions, analogous to the slow knee clonus which I have described ("Med.-Chir. Trans." 1879, p. 289).

It is important to be aware that the inco-ordination may not be equally distributed through the legs. In some patients it is marked in the muscles of the hips and knees, and the legs are raised too high, and brought down too suddenly. In other cases this characteristic is wanting; the inco-ordination affects chiefly the muscles of the feet, and causes unsteadiness of gait and irregular action of the foot-muscles in standing, or in movement of the feet, very conspicuous when they are uncovered.

Controlling Functions—We may consider next the controlling functions of the cord; and first, the influence which it exercises over nutrition. The nutrition of the limbs, etc., is, to a considerable extent, under the influence of the cord; that of the muscles, and probably also of the bones and joints, through the anterior nerve-roots; that of the skin probably through the posterior.

Muscular Nutrition.—For diagnostic purposes the most important is the influence on the nutrition of the muscles. The path of the influence is the motor fibres in the anterior roots and nerves. Changes in the nutrition of the muscles, which are not due to local influence, depend on changes in the nutrition of the motor nerve-fibres. But most, perhaps all, motor fibres are the prolonged processes of the motor nerve-cells, and may be regarded as parts of the nerve-cells, sharing all changes in their nutrition. The nerve-fibres are excitable by electricity, and changes in their nutrition are accompanied by changes in their excitability. By the use of electricity we are able to ascertain their state of nutrition, and thus to learn what is the condition of the nerve-cells in the cord, provided there is no disease separating the part of the fibre tested from the influence of the cells. Hence the value of electricity in the diagnosis of the spinal cord.

In a normal state, if you apply either the faradaic or the voltaic current to a motor nerve, there occurs, as you doubtless know, a contraction in the muscles, continuous when the faradaic current is applied, but, if the voltaic current is used, occurring only when the current commences or ceases to pass—*i.e.*, when the circuit is "made or broken." In proportion as the nutrition of the nerve fibres is impaired, their excitability is lowered; a stronger current of each kind is required to excite them and cause contraction in the muscles they supply. When their nutrition is much impaired—*i.e.*, when the fibres are "degenerated"—no contraction can be obtained even with the strongest currents.

The changes in the excitability of the muscles are less simple because in them there are two excitable structures—the terminations of the nerves, and the muscular fibres themselves. Of these the nerve-fibres are the more sensitive to faradization, and the faradaic stimulation of a muscle under normal circumstances is by means of these motor nerve-endings. Thus we find that its excitability corresponds in degree to that of the motor nerve supplying it. The muscular fibres themselves are, even in the normal state, less sensitive to faradization than the nerve, apparently because they are incapable of ready response to a stimulus so very short in duration as are the shocks of which the faradaic current consists. The proof of this consists in the fact that under the influence of curara, which is believed to remove the excitability of the terminations of the motor nerves, the muscle requires a stronger faradaic current to stimulate it than in the normal state. But under these circumstances the slowly interrupted voltaic current stimulates the muscle as readily as in the normal state, a contraction occurring when the circuit is completed or broken—distinctly slower than that which occurs when the nerve-fibres are intact, and hence almost certainly due to the stimulation of the protoplasm of the muscular fibres themselves. The fact that, under normal circumstances, the contraction which is caused by the voltaic current is as quick as that caused by the faradaic shock, is ground for believing that, in health, the voltaic, as well as the faradaic current, causes the muscle to contract chiefly by exciting the motor nerve-endings. When the motor nerve is degenerated, and will not respond to faradaic or voltaic stimulation, the muscle also loses all its power of response to the former. Apparently the nerve degeneration is accompanied by changes in the nutrition of the muscular fibre, by which any power of response to faradization, which it possessed in the normal state, is lost. But the response to the voltaic current remains, and becomes quickly more ready than in health, probably in consequence of some nutritive changes which develop what the older pathologists called, truly enough, "irritable weakness." Moreover, there may commonly be observed a change in the readiness of response to a certain mode of stimulation with voltaism—a qualitative change, as it is termed. In health, the first contraction to occur, on gradually increasing the strength of the current, is at the negative pole when the circuit is closed, and a stronger current is required before contraction occurs at the positive pole. But, in the morbid state we are discussing, contraction may occur at the positive pole as readily as, or even more readily than, at the negative. This condition, then—faradaic irritability lost, voltaic irritability increased and often changed in quality—is termed the "degenerative reaction," because it occurs when the nerve-fibres are degenerated; and if we test *them* we shall find no response to any stimulus, voltaic or faradaic. It occurs when the nerves are separated from their motor nerve-cells, and if no such separation exists it indicates an acute degenerative change in those nerve-cells. It is well seen in acute myelitis of the anterior cornua (as infantile palsy).

But the motor nerve-cells and fibres often undergo changes in nutrition of a much more chronic character. In this condition the irritability of the fibres is lessened gradually and slowly. The irritability of the intramuscular nerve-endings is also lessened, in the same degree as that of the nerve-trunks, and we have a diminution to both faradization and voltaism. The nutrition of the muscular fibres is slowly, gradually impaired; and when the nerve-fibres are much affected the muscular fibres are also. There is no stage in which the nerve-fibre irritability is lost, and the muscle-fibre irritability retained; hence there is no condition of lost faradaic and increased voltaic irritability such as characterizes the degenerative reaction just described. Irritability is changed to the one form of stimulus just as to the other. This form of change is seen in many very chronic spinal affections, and especially when the nerve-cells suffer, not in consequence of disease primary in them, but as a result of degeneration or irritation spreading to them from above. It is seen, for instance, in the wasting which occurs sometimes in hemiplegic limbs. Between these two forms there are intermediate conditions, especially in cases of subacute disease of the anterior cornua.* In these the (faradaic) irritability of the nerve-fibres may be lost in greater degree than the (voltaic) irritability of the muscular fibres.

Frequently the lowered irritability of degeneration in the nerves is preceded by a slight increase of irritability, very transient when the degeneration is acute, of longer duration when the degeneration is of the slower variety just noticed. Thus, in the early wasting of hemiplegia, increased irritability may be found, slowly yielding to diminution. In some morbid states, again, in which the change of nutrition in the cells and fibres is extremely slight, an increase may alone be discovered. I have found such an increase, for instance, in diseases regarded as functional, as paralysis agitans and chorea, and it is an interesting proof of the molecular changes which underlie, or result from, functional maladies.

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* The various changes in irritability have been thought to indicate the existence and various affections of separate centres for the nutrition of the nerves and muscles, apart from, though acting through, the motor nerve-cells. Remembering that the nerves and muscles contain fibres which suffer in different degrees, the phenomena at present ascertained may, I believe, all be explained on the simpler principle stated in the text, without the assumption of these special centres.

Not uncommonly, in chronic degenerative cases, a form of reaction to faradization is met with, which has not, as far as I know, been described. If the interruptions to this current are very rapid, a contraction occurs when the current commences and ceases to pass, much stronger than that which persists while it is passing. The response thus resembles that obtained with the voltaic current. If the interruptions are less rapid, the contraction is continuous. Even in health, rapidity of interruption lessens readiness of response; and, in this condition, the degenerative changes render the nerve still less sensitive to a rapidly interrupted current (or what is the same thing, to shocks rapidly succeeding one another). The first shock and the last stimulate the nerve more than those which intervene. The indication is that there is chronic degeneration.

In employing electricity as a means of diagnosis, at least one of the electrodes should be small, so as to be able to concentrate the current on a single muscle. Great care must be taken to place these electrodes on corresponding points on the two sides. It is convenient to be able to interrupt the current at the battery, so that the effect of the passage of the current may not be obscured by the mechanical effect of the application of the electrode. In Stöhrer's faradaic battery this can readily be done by pressing the hammer with the third finger, while the rod graduating the current is raised or lowered with the thumb and first finger. By moving the hammer slowly with the finger, we may employ the isolated faradaic shock (the current consisting of a rapid succession of shocks). The isolated shock is often useful, because it is much less painful than the rapid series of shocks, and is especially convenient in the examination of children. A mechanical interrupter is essential in the case of the voltaic battery, in which the stimulation only occurs when the circuit is made and broken; and no battery is suited for use for diagnostic purposes which does not possess such a means of interruption.

In examining muscles and nerves we avail ourselves, whenever we can, of the opposite side for comparison, and when we cannot, we must, if there is any doubt, compare the results obtained with those yielded by a healthy individual. Two conditions may be tested—first, the lowest strength of stimulus to which the muscle or nerve will respond; and secondly, the relative degree of response to a stronger current. The former is the more important, but has perhaps been insisted on too exclusively, for the latter is important also. If a few fibres of a nerve are healthy, and the others are degenerated, contraction may occur with as weak a stimulus as in the healthy nerve; but if the current be made a little stronger, the contraction in the diseased part may remain the same when that on the healthy side is energetic. Both irritability and power, therefore, should be noted.

The nutrition of bones and joints also probably depends on the anterior grey matter, but the influence is shown chiefly by the effect of disease in retarding the growth of bones. Now and then in posterior sclerosis (locomotor ataxy), a painless joint affection and brittleness of bones came on late in the disease. The fact is important; its explanation uncertain.

The nutrition of the skin and subcutaneous tissues depends upon nerves which have their course in the posterior, sensory, roots, but whether there are special trophic fibres is unknown, and the centre on which the influence depends is also unknown. It is doubtful whether simple loss of the function of the posterior roots leads directly to lesions of nutrition. These may result indirectly in this condition; the anæsthesia deprives the patient of sensory information when change of posture is required to prevent damage from pressure. Occasionally, however, sloughing and vesication of the skin occur with extreme readiness, on the least local disturbance, and even with none. This is the case when the lesion is irritative in character, especially in destruction of the cord at the level from which the sensory nerves to the part proceed, and sometimes in disease higher up, as in some cases of acute myelitis.

Micturition and Defæcation.—The spinal cord possesses centres, situated in the lumbar enlargement, which preside over the action of the bladder and rectum. They are probably complex reflex centres: that for the sphincter ani is the more simple, but the system of action of each is probably the same. In the wall of each viscus we have muscular fibres to expel the contents, and at the mouth a sphincter to prevent their continuous evacuation. Fæces or air in the rectum, or urine in the bladder, excites the lumbar centre, and causes contraction in the wall and relaxation of the sphincter. This process may be, to a considerable extent, influenced by the will, although we are still ignorant of the precise mode in which the voluntary influence is exerted. But if the volitional path in the cord is damaged above the lumbar centres, the will can no longer influence the reflex processes; as soon as fæces irritate the rectum, they are expelled by the reflex mechanism; as soon as a sufficient quantity of urine accumulates in the bladder, a reflex contraction of the detrusor and relaxation of the sphincter cause its escape. If the damage to the cord involves also the sensory tract, the patient is unconscious of the process. If the sensory tract is unaffected, the patient is aware of the action of the bladder or bowel, but cannot control it. It is often said that there is permanent relaxation of the sphincters, but this is true only when the lumbar centres are inactive or destroyed. In this condition, evacuation occurs as soon as fæces or urine enter: the urine escapes continuously, instead of being expelled at intervals. The condition is less obvious in the case of the rectum, because there is not such continuous entrance of fæces into the rectum as there is of urine into the bladder. We may, however, distinguish between the two states of the rectum by the introduction of the finger. If the lumbar centre is inactive, there is a momentary contraction, due to local stimulation of the sphincter, and then permanent relaxation. If, however, the reflex centre and motor nerves from it are intact, the introduction of the finger is followed, first by relaxation, and then by gentle, firm, tonic contraction. I have verified this by introducing an india-rubber cylinder instead of the finger, and registering the pressure on the cylinder by connecting it with a recording apparatus, and have found that the relaxation is preceded by a very slight, brief contraction, and is followed by unbroken tonic contraction. The relaxation may also be readily produced by any impression on the mucous membrane of the rectum above the sphincter.

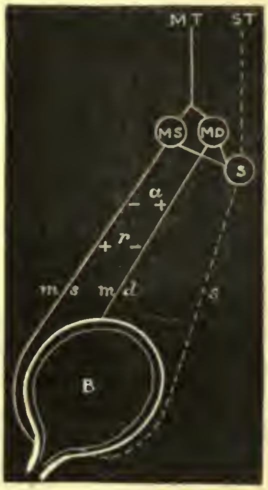

FIG. 13.—DIAGRAM SHOWING PROBABLE PLAN OF THE CENTRE FOR MICTURITION.

MT motor tract, ST sensory tract in the spinal cord; MS centre and *ms* motor nerve for sphincter; MD centre, and *md* motor nerve for detrusor; *s*, afferent nerve from mucous membrane to S, sensory portion of centre; B, bladder. At *r*, the condition during rest is indicated, the sphincter centre in action, the detrusor centre not acting. At *a*, the condition during action is indicated, the sphincter centre inhibited, the detrusor centre acting.

In cases of gradual disease we may often trace the gradual loss of voluntary power over the process of micturition. In some cases this loss of power appears to be manifested as an inability, not to restrain, but to excite the action of the centre, and we have then a tendency to retention. Many conditions can be best understood by assuming that the motor centre really consists of two parts—one (MS, Fig. 13) maintaining the contraction of the sphincter, the other (MD) exciting the contraction of the detrusor fibres, and that these two parts are antagonistic: when one acts, the other is inhibited. Thus, in normal rest, the sphincter centre is active, the detrusor at rest. Action is produced by a conjoint afferent impulse from the bladder and voluntary impulse from the brain. Then the detrusor centre acts, and the sphincter centre ceases to act. If voluntary power is impaired, the afferent impulse from the bladder may be insufficient, and then there is retention, or in other such cases the motor centre may yield too readily to the afferent impulse, and there is reflex incontinence.

Sexual Functions.—The conditions of the sexual organs depend on the integrity of the reflex loop to and from a special centre, also situated in the lumbar enlargement, but the due action of this centre depends on cerebral (psychical) as well as reflex influences. Disease of the centre, or of the nerves leading to or from it, abolishes sexual action. The sexual reflex is, however, one of the superficial reflexes, the excitation being from the skin, and it shares the condition of the superficial rather than of the deep reflexes.

The centre is probably double, and its action is impaired by interference with either half. When, by disease higher up, the connection with the psychical centres is interrupted, the sexual act cannot be perfectly performed. If the path from the controlling centre (p. 19) is unimpaired, the reflex sexual processes are not in excess, may even be diminished; but if the path from this controlling centre is also interrupted, the reflex sexual processes are in excess like the other superficial reflexes, and priapism results. If the reflex centre is partially diseased, the sexual act is imperfectly performed.

The sexual centre is probably near that for the cremaster reflex, and from the latter we may, in some cases, gain information respecting the probable condition of the sexual centre, and in conjunction with the conditions just mentioned, of the sexual power. For instance, in locomotor ataxy, I have never seen the cremaster reflex lost* without sexual power being lost or lessened also; and in a case of this disease, in which all the superficial reflexes, including the cremaster, were in marked excess, there existed satyriasis. Sexual power may, however, be lost before the cremaster reflex, perhaps because more readily impaired. Again, in a patient with extensive disease of the lower and middle part of the lumbar enlargement, greatest on the left side, in whom the cremaster reflex was present on the right side and lost on the left, sexual power was partially but not wholly lost.

Vaso-motor Centres.—The centres in the cord which influence the sympathetic and vaso-motor system of nerves are frequently affected in disease, and altered temperature, vascularity, and perspiration of limb result. In disease of the upper part of the cervical enlargement, especially in sudden lesions, hyperpyrexia may occur. But these symptoms at present are of little diagnostic importance, except when the disease is in the cervical region, and the vaso-motor change is conspicuous in the face. Then, if one side is affected, unilateral sweating and flushing are conspicuous, and are due to the fact that sympathetic fibres for the head arise in, or pass through, the cervical cord. In the same cases, the movements of the iris are impaired: irritation of the cervical origin of the sympathetic causing spasm of the radiating fibres (dilatation), paralysis of the sympathetic causing their relaxation (contraction of the pupil). In many degenerative diseases of the cord the reflex action of the pupil to light is lost, the pupils being usually, but not always, small, but (as Argyll Robertson first showed) the pupils almost always contract if an effort of accommodation is made. The reflex action is lost, but the associated action remains. It is not certain that these phenomena depend directly on the disease of the cord; they are perhaps due to an associated degeneration in the centres for the movements of the iris in the upper part of the pons. In many cases the latter explanation is the more probable.

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* It must be remembered that the cremaster reflex is sometimes absent, in adults, apart from disease. The statement in the text applies to the cases in which there was such a general absence of the superficial reflexes as suggested a pathological loss.

In this survey of the more important functions of the cord, and their derangement, we have passed in review the chief symptoms which guide us in diagnosis. One or two others, however, remain.

Pain, referred to the spine, is occasionally present in organic disease of the cord, but is more frequent in disease originating in the meninges or bones. But the frequency with which spinal pain is present in abdominal, especially gastric, disease, and in neuralgic affections, lessens its diagnostic value when it exists alone. It is probably no exaggeration to say that of one hundred patients who complain of spinal pain, in ninety-nine there is no spinal disease. Moreover, in cases of organic disease, pain is far less frequent when the disease begins in the cord, than when it commences in the protecting structures, membranes, or bones. In meningitis, acute or chronic, spinal pain is frequent, and in organic disease of the bones of the vertebral column it is an almost constant symptom, and is combined with local tenderness. The same combination of local pain and tenderness is seen, however, in some cases of neuralgic pain, "rachialgia." The distinction between the two is, that in organic disease there are indications either of displacement of the vertebræ or of changes in the cord.

A still more important group of pains are those which are referred to the parts to which the sensory nerves are distributed, and have hence been termed "excentric pains." They are due to the irritation of the posterior nerve-roots, in their passage through the vertebral foramina, through the membranes, or through the posterior columns of the cord. Other similar pains are due, apparently, in some cases, to irritation of the sensory conducting tract higher up the cord.* They may be dull pains, singularly resembling rheumatism, and constantly mistaken for rheumatism by the patients themselves and their medical attendants. The mistake is the more easily made, because other symptoms suggestive of spinal disease may be inconspicuous, and the rheumatoid pains, in acute cases, may be accompanied by febrile symptoms, and in chronic cases, may be influenced by weather. In all cases, persistent rheumatic pains in the limbs should excite a suspicion of spinal disease, and watch should be kept for such symptoms as local loss of power, or alterations in reflex action. In other cases they are sharp darting pains, "like a flash of lightning and gone again," as they are often described by patients with locomotor ataxy, in which disease they are very frequent. The position in which these various excentric pains are felt—legs, trunk, or arms—depends (when the nerve-roots are irritated) upon the seat of the disease—in the lumbar, dorsal, or cervical regions of the cord. Occasionally the irritation is felt, not as a sharp pain, but as a painful sense of tightness, as if a band were tied tightly around the limb or trunk—the "girdle-pain," as it is called. When there is transverse damage to the cord, at the lowest part of the healthy region there is a state of irritation of the sensory nerves, and this irritation (referred to the nerve-endings) causes the girdle-pain. When the nerve-roots are irritated by disease of the vertebræ, the pain is very intense, and is especially increased by movement. In cancer of the bodies of the vertebræ this pain is so severe that the disease has received the name of "paraplegia dolorosa."

Whenever there are excentric pains there may be increased or diminished sensitiveness in the part to which the pains are referred. Spontaneous sensations are also common, the various feelings comprehended under the terms "numbness," "pins and needles," "furriness," "formication," and the like. The significance of these, as suggestive of central disease, is commonly recognized, and of their more precise indications we are still ignorant.

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* The latest physiological researches seem to show that the conducting tracts in the cord are not, as is commonly taught, entirely insensitive to local stimulation.

Spasm.—Muscular spasm is conspicuous in many cases of disease of the spinal cord. It depends essentially on an excessive action of the motor centres, especially the reflex centres. Primarily, perhaps, it is due to "diminished resistance" within them, but ultimately the functional action (and underlying nutrition) of these centres seems to be permanently altered. The motor centres are, as we have seen, not only parts of the reflex centres, but also the terminations of the path of voluntary impulse. Hence spinal spasm may be excited by peripheral impressions, or by attempts at voluntary motion. In some cases paroxysms appear to come on without excitation, especially during sleep. In sleep, however, the reflex action of the cord is very ready, and it is difficult to exclude slight reflex stimulation. As an acute symptom, spasm is almost confined to meningitis, and to some very rare forms of functional irritation. Meningitis apparently causes a direct intensification of the action of the reflex centres. In chronic organic disease, spasm is usually a late symptom of gradual development, and then its reflex character may often be distinctly traced. It occurs in cases in which the reflexes, and especially the deep reflexes, are in excess, *i.e.*, when the reflex loops are entire, and the cerebral influence is lessened by disease higher up, involving the lateral columns (motor path). It does not occur, however, immediately, but after one or more weeks, and so is probably due, not to the mere withdrawal of cerebral influences, but to the occurrence of descending degeneration in the lateral columns. We may observe in such cases the gradual development, first of excess in the deep reflex actions (increased knee reflex, ankle clonus), and then of occasional slight "stiffness," and ultimately of paroxysmal spasm and rigidity, until the degree known as "spasmodic paraplegia," or "spastic paraplegia," is reached. Any peripheral impression, superficial or deep—pinching the skin, for instance, or sudden muscular tension—

will then excite spasm. The attempt to elicit the ankle clonus may cause such muscular rigidity that no clonus can be obtained. In most cases the spasm is extensor in character, and evidently depends on the reflex mechanism which assists in maintaining extension of the legs in the erect posture. In health, when we stand, the muscles are in a state of balanced contraction, largely reflex, the afferent impulses being derived from muscles and joints. In spastic paraplegia a similar but more intense extensor contraction is excited by the same posture of the limb. Flexed, it may be supple, but extend it passively, and as soon as it is straight the muscles become rigid, and it cannot again be flexed except by considerable force. It is just as when a clasp-knife is opened, as soon as the blade is fully extended it becomes rigid. So this has been called "clasp-knife rigidity." Frequently the spasm fixes for the time both legs to the pelvis, so that if one leg is lifted from the bed the other rises with it. The same extensor spasm occurs when the patient attempts to stand, and it often enables a patient to remain erect whose voluntary power would be insufficient for him to do so were he not aided by the spasm.

In some cases, especially during sleep, flexor spasm predominates, and the hip and knee joints become strongly flexed. On what the difference in the form of spasm depends we do not yet know. Spasm, especially flexor spasm, was formerly regarded as evidence of "chronic meningitis," because acute meningitis is accompanied by spasm. In many of these cases, however, there is no other evidence of meningeal disease.

Occasionally spasm occurs in violent paroxysms, first tonic, and then clonic, excited by slight peripheral impressions, and in some cases apparently spontaneous—the "spinal epilepsy" of Brown-Séquard. I think, however, that the resemblance to an epileptic paroxysm is superficial, and that the quick clonic spasm depends on precisely the same conditions as the ankle clonus. The peripheral impression excites tonic spasm; as this is passing off, the tension on the imperfectly relaxed muscle is sufficient to develop clonic contractions, just as does the passive tension in the ordinary method of obtaining the ankle clonus, and so we have a series of quick clonic contractions succeeding tonic spasm. The effect is most conspicuous in the quadriceps extensor of the knee. Sometimes the initial tonic spasm is slight, and the spasm consists entirely of clonic spasm.*

Thus these spasmodic phenomena indicate integrity of the reflex loops and functional over-activity of the reflex centres. This over-action may be, in acute cases, the result of meningeal inflammation; in chronic cases it is the effect of disease above, in the lateral columns, the degeneration of which extends down to the lower centres, but does not, apparently, invade them. The gradual development of the over-action indicates that it is the result, in most cases, of changes consequent on this degeneration. In what those changes consist we do not know. The excessive reflex action gradually leads to what may be termed, if the expression is permissible, a functional hypertrophy of the centres, causing persistent and extreme spasm. How far this is due to a condition of "irritation" propagated from above, we do not know, but I do not think that the assumption is necessary, or justified by the facts.

Simple rigidity of muscles, varying too little to be termed spasm, occurs also in some forms of disease of the cord, especially in cases of muscular atrophy (degeneration of the anterior cornua), and is due (according to Charcot) to simultaneous degeneration in the lateral columns. Fixed contraction of muscles occurs also in the antagonists of paralysed muscles, but now and then as a result of over-action from central disease. As a consequence of this the knees may become flexed or the heels drawn up. The latter form of rigidity, dependent on a primary over-action, is always associated with more or less general spasm in the limb—an important distinction from the condition in which such shortening is due to the paralysis of the opponents of the contracted muscles. Persistent contraction of the gastrocnemii, as part of "spastic paraplegia," is sometimes seen in adults, but is more common in children.

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* It may be well to state the grounds on which this opinion is based. In cases in which the superficial and deep reflexes are in great excess, and the ankle clonus readily excited, a touch on the sole may cause general tonic spasm, and as this is passing off there may be a series of clonic contractions in the calf muscles, exactly similar to the ankle clonus, no passive flexion of the ankle having been made. They evidently result from the effect, on the relaxing muscle, of the tension produced by the preceding clonic contraction. Again, in a patient now under my care, when tonic spasm has been excited in a leg, sudden brief tension on the extensors of the knee excites a series of quick clonic contractions in these muscles, which continue for a few seconds, the limb being left alone. The spasm exactly resembles that of ordinary spinal epilepsy.

It is necessary to mention that the term "spinal epilepsy" has been applied in France to the ankle clonus.

III.—INDICATIONS OF POSITION OF DISEASE: ANATOMICAL DIAGNOSIS.

We may now consider, briefly, how the symptoms which we have studied are grouped in diseases of different regions of the cord. The various symptoms, and their significance, have already been considered in detail, so that it is necessary only to mention them. Some lesions of the cord affect certain structures (white columns or grey matter) in a considerable vertical extent, the other structures being normal. Such affections have been called "system diseases." Others, again, are very limited in their vertical extent, and have been termed "focal" lesions. The latter may be limited to one structure, or may extend through a considerable transverse extent, even through the whole thickness of the cord—"total transverse lesions." The lesions which affect certain structures only, whether extensive system diseases or limited focal diseases, are called "partial lesions," and it is convenient to commence with these.

1. *Antero-lateral White Columns.*—Disease of the antero-lateral white columns causes loss of voluntary power below the lesion, descending degeneration in the pyramidal tracts, and over-action of the lower centres. This over-action may be manifested only as excessive knee reflex and developed ankle clonus, or it may increase from this to spasm and rigidity—spastic or spasmodic paraplegia. There is no wasting unless the degeneration extends from the lateral

columns to the anterior cornua. Then we have a combination of spasm and wasting in which, if the cornual degeneration proceeds, the spasm and rigidity may lessen as the cornua suffer. In disease limited to these columns (at any rate, when the disease is limited to the pyramidal tracts) there is no loss of sensation or inco-ordination. These symptoms of "spasmodic paraplegia" may arise from a primary degeneration in the lateral columns, limited thereto; but such cases are extremely rare, and in the majority the disease is a focal lesion more or less extensive at some level in the dorsal or cervical cord, and the degeneration in the lateral columns is secondary. The evidence of the latter form is afforded (1) by the circumstance that the symptoms came on, in the first instance, suddenly or rapidly, primary sclerosis being always gradual in onset, and a lesion which occurs in short time is always "focal"; (2) the evidence which may generally be discovered that there has been at some time, or is in some region, damage which extends beyond the lateral columns. The proof of this is the implication of sensation, or the interference, at the level of the lesion, with the central functions of the cord. We are only justified in diagnosing a primary sclerosis of the lateral columns when we can find no such evidence or history of a wider focal lesion, and when the affection came on very gradually. We must remember also that descending lateral sclerosis, with secondary spasmodic phenomena in the limbs, may even result from damage to the motor tracts above their decussation—in the medulla, the pons, or the motor parts of the cerebral hemispheres. It occasionally results from bilateral damage to the surface of the brain during difficult birth.

Certain lesions may damage the motor tracts slightly, and impair conduction in a peculiar way, rendering it apparently unequal in different fibres. As a consequence, the muscular action is unequal in different muscles, and instead of a balanced co-ordinated movement, we have an unbalanced jerky movement. This is seen especially when irregular islets of sclerosis affect the cord—disseminated or insular sclerosis; and according to the researches of Charcot it appears that this irregular conduction is the result of the unequal wasting of the medullary sheaths, the axis cylinders remaining. A precisely similar symptom may result from pressure on the motor tract—as by a growth. Not rarely this "disseminated" or "insular" sclerosis, in one region, is combined with a system-degeneration in another. An occasional combination, for instance, is the jerking movement (from cervical insular sclerosis) in the arms, and weakness with spasm (from lumbar lateral sclerosis) in the legs. In such cases it is probable that the lateral sclerosis is simply "descending," the result of the damage to the pyramidal tract by the insular sclerosis. It must be remembered that insular sclerosis may cause merely loss of power, and equal, not irregular, impairment of conduction, especially when it occurs in the dorsal region. In such a case we may be unable to distinguish its symptoms from those of a diffuse, widely-spread degeneration.

2. *Posterior Columns.*—In disease of the posterior columns there is interference with co-ordination without loss of power; excentric pains, impaired sensation, and diminution of reflex action, in consequence of the implication of the sensory roots. All these symptoms depend on disease of the postero-external columns (posterior root-zone). Disease of the postero-median column gives rise to no known symptoms.

The posterior columns may be damaged by any pathological process: they are frequent seats of primary degeneration (sclerosis), the condition which constitutes the common form of locomotor ataxy. The symptoms of this disease usually present the following order — loss of the deep reflexes, pains, inco-ordination, diminution of sensation, loss of sexual power, and of the superficial reflexes, affection of the sphincters, and occasionally interference with the nutrition of bones and joints.

There is no loss of motor power or wasting as long as the disease remains limited to the posterior columns. It may, however, extend forwards into the anterior cornua, causing muscular atrophy and weakness to be conjoined with the ataxy. Or the lateral columns may be affected at the same time as the posterior: we then have weakness as well as ataxy, but no wasting. The disease of the lateral columns causes, as I have just stated, increase of the deep reflexes, and this increase may thus co-exist with inco-ordination, the damage to the posterior roots being perhaps irregular in these cases. Thus we may have the anomaly of ataxy with excess of the knee reflex instead of its loss, and with the front-tap contraction obtainable, and even the ankle clonus.

An important fact to remember regarding the posterior columns is their proneness to degenerate: they recover less readily, and degenerate more readily, than any other part of the cord. A lesion in one spot may set up a degeneration which ultimately involves them in their whole extent. Damage affecting the whole thickness of the cord may pass away from the rest, and persist in the posterior columns, and even spread there. In such a case we have ataxy succeeding loss of power. Movement returns, but without co-ordination.

3. *Anterior Cornua.*—The anterior cornua contain the motor nerve-cells, which, as I have said—(1) influence the nutrition of the motor nerve fibres proceeding from them, and consequently that of the muscles; (2) constitute a link in the path of the voluntary impulse from the brain to the muscles; (3) form part of the reflex loop, probably also of the reflex centre, with which the muscles are connected.

Hence we have, as the result of disease of the anterior cornua—(1) degeneration of the motor nerves and wasting of the muscles; (2) loss of voluntary power, *i.e.*, paralysis of those muscles; (3) interference with, or arrest of, the reflex actions in which these muscles take part.

The extent of these symptoms, whether they are unilateral or bilateral, affect many muscles or few, will depend strictly on the extent of the disease in the spinal cord.

Of the three symptoms, the muscular wasting is incomparably the most important. Paralysis may result from disease elsewhere in the motor tract, *e.g.*, disease of the lateral column higher up. Loss of reflex action may depend on disease elsewhere in the reflex loop, *e.g.*, disease of the sensory fibres in or outside the cord. But muscular wasting is due only to a lesion of the motor cells, or to a lesion of the nerves cutting the muscles off from the influence of these cells. In most cases we are able to exclude the latter (by indications presently to be mentioned): the state of muscular nutrition comes thus to be of the highest importance as indicative of the state of the anterior cornua of the cord. To learn their condition further, we ascertain the electrical excitability of the nerves and muscles, according to the principles already laid down.

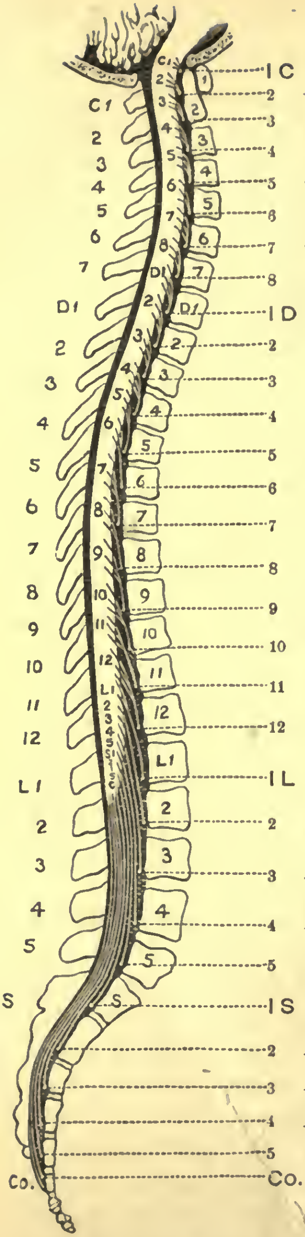
Whenever we find wasting, and infer that there is disease of the anterior cornua, we have next to observe whether the weakness and wasting are proportioned, *i.e.*, whether the weakness is only such as the affection of the grey matter will account for, or whether it is in such excess as to indicate other disease in the motor tract.

Disease of the anterior cornua is often combined with disease of the lateral (pyramidal) columns similar to the descending degeneration. Charcot believes that in these cases the degeneration in the lateral column is primary, its symptom, muscular rigidity, preceding the symptom of the cornual disease, muscular wasting, and he terms the affection "lateral amyotrophic sclerosis." It is possible, however, that this position will need reconsideration, and that the degeneration in the lateral columns is, sometimes at least, secondary to, or simultaneous with, the disease in the cornua. It often spreads, however, beyond the fibres related to the degenerated cornua, and so may cause weakness and spasm in the limbs below the seat of the muscular atrophy. Thus we have wasting in the arms, and weakness with spasm in the legs, and even, as I have seen, wasting in the shoulder muscles, and weakness without wasting in the hands.

A lesion of the anterior cornua never, *per se*, affects sensation. Acute lesions in this situation may, however, disturb adjacent sensory parts (posterior cornu or sensory tract), and so cause "excentric" pains, often rheumatic in character. Actual loss of sensation, with wasting, points, especially if irregular in distribution, to damage to the nerve-roots rather than to the cord itself.

4. *Unilateral Lesions.*—These interfere with the conduction of the motor impulse on the same side as the lesion, and so cause weakness in one side, "hemi-paraplegia" or "spinal hemiplegia," and often descending unilateral sclerosis with its symptoms in the affected leg. Whether there is weakness of the other leg (if the lesion is strictly unilateral) will depend on the number of pyramidal fibres which, in the "direct pyramidal tract," have not decussated at the seat of the lesion; and this varies, as we have seen, in different individuals. It must be remembered, however, that in unilateral lesions, the opposite half of the cord is often slightly damaged, and the symptoms are therefore rarely strictly limited to one leg. Sensation is affected on the opposite side to motion, but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate, but occurs a little above the place at which the nerves enter the cord. Sometimes, however, sensation is affected on the same side as motion. This is often the case when the paralysis affects the leg only, and probably the lesion is, in such cases, so low as to impair the sensory fibres before their decussation.

5. *Total Transverse Lesions.*—A total transverse lesion of the cord, at any level, however limited in vertical extent, separates all parts below the lesion from the brain, and hence, so far as will and perception are concerned, produces the same effect as if the whole of the cord below the lesion were destroyed. A section across the cord in the middle of the cervical enlargement, for instance, paralyses all parts below the neck. Hence the extent of the paralysis indicates only the upward extent of the lesion. This is also indicated by the position of the girdle pain, or zone of hyperæsthesia, which is due to the irritation of the sensory roots in the lowest part of the



MOTOR.

- St.-mastoid
- Trapezius
- Diaphragm
- Serratus
- Shoulder } mus.
- Arm } mus.
- Hand } mus. (ulnar lowest)
- Intercostal Muscles
- Abdominal Muscles
- Flexors, hip
- Extensors, knee
- Adductors } hip
- Abductors } hip
- Extensors(?) } hip
- Flexors, knee (?)
- Muscles of leg moving foot
- Perineal and Anal muscles

SENSORY.

- Neck and Scalp
- Neck and Shoulder
- Shoulder
- Arm
- Hand
- Front of Thorax
- Ensiform area
- Abdomen (Umbilicus 10th)
- Buttock, upper part
- Groin and scrotum (front)
- Thigh
  - outer side
  - front
  - inner side
- Leg, inner side
- Buttock, lower part
- Back of Thigh
- Leg and Foot } except inner part
- Perineum and Anus
- Skin from coccyx to anus

REFLEX.

- Scapular
- Epigastric
- Abdominal
- Cremasteric
- *Knee reflex*
- Gluteal
- *Ankle clonus*
- Plantar

FIG. 14.—DIAGRAM AND TABLE SHOWING THE APPROXIMATE RELATION TO THE SPINAL NERVES OF THE VARIOUS MOTOR, SENSORY, AND REFLEX FUNCTIONS OF THE SPINAL CORD. (*From anatomical and pathological data.*)

upper segment—an important indication when the lesion is in the dorsal region, where the precise limitation of motor weakness may be recognized with difficulty.

It is important, however, to know the symptoms which occur in disease at different levels. These are shown in the accompanying diagram and table (Fig. 14), and may be understood from the following description. The indication of the upper level of the lesion is afforded by the loss of the motor and sensory functions, shown in the first two columns. The lowest nerves supply the anus and perineum. The nerves which supply the skin and muscles of the leg and foot arise from the 1st to the 3rd sacral nerves, and are damaged by a lesion involving the lower part of the lumbar enlargement. We must remember, however, that the skin on the inner side of the leg is not supplied from this source, and so may escape when the outer part of the leg and back of the thigh have lost sensation. In the middle of the lumbar enlargement, we have the nerves arising which enter the lumbo-sacral cord, and these are probably destined for the flexors of the knee, and for the hip muscles which are supplied by the sacral plexus, the gluteal, the quadratus and gemelli, and the skin of the lower part of the gluteal region. These parts then will be paralysed by disease in the middle of the lumbar enlargement, while the muscles and skin in the front of the thigh are unaffected. The latter suffer when the disease affects the upper part of the lumbar enlargement, the origin of the anterior crural (rectus, etc.), and obturator (adductors). The skin on the upper and outer parts of the thigh loses sensibility, with the part adjacent to the scrotum, and in the groin, only when the disease damages the highest part of the lumbar enlargement, from which the first three lumbar nerves arise, and then the flexors of the hip become paralysed. In proportion as the disease is higher in the dorsal region, we have the symptoms ascending higher up the trunk, and marking accurately the height of the lesion by the loss of cutaneous sensibility, and by the impairment—first, of the abdominal muscles, and then of the intercostal muscles. The umbilicus corresponds to the 10th dorsal nerves, and the ensiform area to the 6th and 7th. When the disease reaches the lowest part of the cervical enlargement (the 1st dorsal nerves), we have the first symptoms in the upper extremity; but these are not, as might be expected, in the muscles moving the shoulder joint, but in the hand. The first numbness is complained of in the little finger, and the first weakness is in the intrinsic muscles of the hand. Ascending higher, the symptoms pass up the arm with some uniformity, and without respect to nerve distribution. When the middle of the cervical enlargement is reached (the 5th, 6th, 7th cervical), the shoulder muscles and the serratus magnus become paralysed, and there is general loss of power and sensation and anæsthesia. Above the level of the 6th pair, the trapezius and sterno-mastoid become weakened, for the fibres of the spinal accessory which supply them undoubtedly arise from this part of the cord. At the 4th and 5th cervical the lower part of the neck becomes anæsthetic, and the diaphragm ceases to act. Here our localization might cease, for total transverse lesions at this spot necessarily cause death. But limited lesions may occur higher up, and then we have complete powerlessness of the muscles moving the head, upper part of trapezius and sterno-mastoid, and other muscles attached to the occipital bone, and interference with sensation in the neck and parts of the head, which are not supplied by the 5th nerve.

The extent downwards of the lesion, its vertical extent, is thus not indicated by the impairment of its conducting functions, the motor or sensory paralysis; and to learn *this* we have to examine the functions of the cord as a central organ, and to ascertain how far they are impaired in the paralysed region—to examine especially muscular nutrition and reflex action. The state of muscular nutrition and irritability indicates how far the anterior cornua are impaired, and the latter shows, as I have explained (p. 34), whether they are involved in the primary lesion or are affected secondarily. The relation of the several groups of muscles to the cord is shown in the first column of the table. The integrity of reflex action indicates the integrity of the reflex loops, and the study of the superficial reflexes of the trunk is especially instructive in this respect. The series of reflexes and the relation of each to the cord, is shown in the third column of the table. Excess of superficial reflex action indicates withdrawal of the cerebral controlling influence of the reflex centres, and marked excess of the deep reflexes suggests the existence of a descending degeneration in the lateral columns.

IV.—INDICATIONS OF NATURE OF DISEASE: PATHOLOGICAL DIAGNOSIS.

The last part of our subject remains for consideration—the elements of the pathological diagnosis, by which, having ascertained the seat of the lesion, we endeavour to learn its nature. To do this, we attend, first to the way the symptoms come on and develope; secondly, to the position and distribution of the lesion; thirdly, to any casual or associated conditions which may be present.

We may group the primary morbid states into the following forms:—

(*a*) Vascular lesions; rupture of vessels, causing hæmorrhage; occlusion of vessels, from thrombosis or embolism (the latter being very rare).

(*b*) Inflammation; "myelitis," acute or chronic, causing softening. It is common to call all forms of softening "myelitis"; we do not yet know how far they are originally inflammatory, or are set up, as in the brain, by vascular occlusion.

(*c*) Degeneration, or "sclerosis," in which the nerve-fibres waste, and the connective tissue (neuroglia) overgrows.

The term "sclerosis" is inaccurate etymologically, since the part altered by increase of connective tissue elements is often softer than normal, but the term seems to be firmly rooted. In some cases the change appears to commence in the nerve-fibres, in others in the connective tissue. Some forms of degeneration pass by gradations into chronic inflammation (here as elsewhere), and the term "chronic myelitis" is sometimes applied to the slow degenerative forms. On the other hand, the condition of "sclerosis" may result from inflammation. The term is thus used in two senses, to indicate a pathological process, and a pathological condition which may result from more than one morbid change. Here the term will be used, when unqualified, to designate the process.

(*d*) Pressure from without, by inflammatory swelling of meninges, or by displaced bones, or by growths.

(*e*) Growths in the cord itself.

I. We have first to consider how far these several lesions can be distinguished by their onset, *i.e.*, by the time occupied in their development to a considerable degree of intensity. According to this, we may divide them into six classes: those in which the onset is *sudden*, instantaneous or nearly so; *acute*, occupying a few hours to a few days; *subacute*, developing in one to four weeks; *subchronic*, in one to two months; and lastly, the *chronic* cases, which may be divided into those occupying two to six months, and those occupying six months and upwards in their onset.

I have endeavoured to show the common relation of the lesions to these several courses in the following table:—

	Onset.	
	Sudden (*few minutes*)	Vascular lesions.
	Acute (*few hours or days*)	
Pressure or Growths	Subacute (*one to four weeks*)	Inflammation (myelitis).
	Subchronic (*one to two months*)	
	Chronic (*two to six months*)	Degeneration (sclerosis).
	Very Chronic (*six months and upwards*)	

A lesion of sudden occurrence, developing symptoms in the course of a few minutes, is always vascular; commonly hæmorrhage, perhaps sometimes vascular obstruction. But a vascular lesion may occupy a somewhat longer time in development—a few hours or days. In acute and subacute inflammation the symptoms come on in the course of a few hours, a few days, or a week or two. Chronic inflammation occupies from a few weeks to a few months. Degeneration, in which there is no adequate evidence of any inflammatory process, occupies many months, or it may be years. The symptoms produced by growths or simple pressure (traumatic causes excluded) are never sudden or very acute, and rarely, if ever, very chronic, the time occupied by the development of the symptoms varying, according to the nature of the cause, from a fortnight to six months.

It is necessary to consider, however, not merely the whole time occupied by the development of the disease, but also the uniformity of its course. Two or more forms of lesion may concur. An initial myelitis, for instance, may lead to a secondary degeneration; and, on the other hand, in degenerated tissues sudden vascular lesions occasionally occur. So the whole course of the disease must be ascertained before an inference is drawn.

The onset and course of the symptoms thus sometimes enable us to decide at once that a lesion is of a given character, as that one which occurs instantly is vascular, or that one which takes years for its development is degenerative. More frequently they enable us to exclude certain morbid processes, and to restrict the possible lesion to two or three forms. For instance, a lesion which comes on in the course of a few hours must be either vascular or inflammatory. Between these we have to decide by attention to other indications.

II. In actual diagnosis it is convenient to consider next the indication afforded by the position and distribution of the disease. We consider what diseases occur in this situation, and then which of them have the mode of onset which has been ascertained. As I said at the outset, this indication of seat of lesion is never to be employed alone—never, except in subordination to a careful study of the mode of onset and course.

The grey matter of the cord is the most frequent seat of hæmorrhage. Either grey or white substance may be the seat of inflammation or of degeneration. Pressure or growths usually first affect the white columns, but may afterwards involve the grey matter.

The affections called "system-diseases," in which one system of structure is affected through a wide vertical extent of the cord, are commonly degenerative in nature: such are lateral sclerosis; posterior sclerosis (locomotor ataxy), the change in the anterior cornua which leads to progressive muscular atrophy (anterior cornual degeneration). These processes *probably* begin in the nerve elements. On the other hand, lesions which have a limited vertical extent —"focal lesions"—are commonly the result of processes which may be either acute or chronic, but begin outside the nerve elements, in the connective tissue, vessels, etc. Such are hæmorrhages, foci of myelitis, spots of "insular" sclerosis, growths, and pressure from without.

But this distinction cannot be employed except after due consideration of the mode of onset. Scattered acute focal lesions, for instance, may be widely distributed in the same structure, and produce symptoms limited to certain functions, but of wide extent, and simulating—indeed, constituting—a "system-disease." Thus I have seen sub-acute symmetrical myelitis of the anterior cornua in the lumbar and cervical enlargement cause paralysis and atrophy in all four extremities, the upper parts of the limbs being normal. Again, a small focal lesion may be limited to one structure, and cause symptoms confined to one function. Thus we may have an anterior cornual myelitis, or a columnal myelitis, lateral or posterior, giving rise to limited symptoms—focal muscular atrophy, unilateral paralysis, or local ataxy. Lastly, many "focal lesions" may give rise to secondary system-degenerations. A focus of myelitis in one lateral column may cause descending degeneration in the whole lateral column below, with its attendant spasmodic symptoms. Indeed, so true is this, that, as we have seen, lateral sclerosis is one of the rarest of primary lesions—is almost always secondary to a limited focal lesion. In all these cases, however, attention to the mode of onset will prevent error.

The combination of mode of onset with seat of lesion sometimes helps us in a more direct manner, especially in the case of growths and compression. The characteristics are their limited vertical extent, gradual onset, and slow invasion of parts adjacent to that first affected, on the same level; one leg, for instance, is affected, and then the other.

There is a rare form of paralysis, in which the functions of the cord are progressively impaired from below upwards, until, in the course of a few days, death results from interference with respiration. In these cases of "acute ascending paralysis," as they are called, no lesion of the cord has usually been discovered, and their exact nature is unknown. In one case of the kind, I found hæmorrhage outside the dura mater compressing the cord. It is possible that this lesion has been overlooked in other cases, on account of the amount of blood always found in this situation in consequence of the division of vessels in opening the canal, the vessels being always gorged from the mode of death and position of the body.

III. The last element in the pathological diagnosis is the detection of any influence which can be regarded as the cause of the disease in the spinal cord, or any associated condition which may indicate an active morbid process. We have seen that the mode of onset may help us to limit the possible disease to certain forms of lesion: the distribution of the affection may render it probable that it is one or other of these forms; and the detection of a cause of disease of the spinal cord, and the knowledge of the lesions which that cause produces, may help us to fix the nature of the lesion still further. It is important, therefore, in diagnosis to be aware of the several effects of the common causes of spinal disease.

1. *The State of the Vascular System.*—The conditions which favour hæmorrhage are of far less diagnostic value with regard to the spinal cord than with regard to the brain. Conditions of mechanical congestion—heart disease, emphysema, etc.—favour degenerative changes and also, probably, thrombosis. The state of the vascular system which is associated with chronic kidney disease undoubtedly favours degenerative changes in the cord, the occurrence of which has been demonstrated by Sir William Gull and Dr. Sutton.

2. *Scrofula* commonly causes spinal disease by leading to disease of the bones of the spinal column; and the evidence of this, local tenderness or irregularity in the vertebral spines, or actual curvature, is of the highest diagnostic importance, and careful and repeated examination of the bones should never be neglected in cases of obscure spinal disease. There is, perhaps, no error in diagnosis which is more frequently made, or which results in graver errors in treatment, than the non-recognition of disease of the spinal bones. It is important to remember that the damage to the cord may occur before the signs of bone disease are distinct: hence the importance of *repeated* examinations.

In bone disease the cord suffers in at least four different ways:—(*a*) By pressure from the inflammatory swelling of the bone without curvature. The effects of the pressure may lessen as the curvature comes on. (*b*) By pressure in consequence of the displacement, the bony canal being narrowed by the angular projection of the bodies over which the cord is stretched. (*c*) By secondary chronic inflammation, with thickening, of the dura mater (pachymeningitis), compressing the cord. (*d*) By the extension of an acute inflammation from the bone through the membranes to the cord. Hence we cannot, because we find evidence of bone disease, immediately conclude that the cord is pressed upon by the displaced bone. We must investigate the mode of onset of the symptoms and their character, and infer from these the character of the disease of the cord according to the rules now given.

In recognizing bone disease it must be remembered that not only may there be no angular curvature until long after the cord has suffered, but there may not even be irregularity of the vertebral spines. There is often pain and local deep tenderness to be elicited. In the resulting paraplegia the excess of the superficial reflexes is often an early and conspicuous symptom, and spots of anæsthesia at the level of the bone disease, due to pressure on nerves, may sometimes be found, and give important help in diagnosis.

3. *Syphilis.*—The methods by which syphilis causes disease of the cord, which are universally recognized, are—

(*a*) The growth of syphilomata springing from the connective tissue, the membranes or tissue in the fissures, and invading the cord. In these cases we have symptoms varying in character according to the position of the growth, and similar to those produced by other limited lesions, but always of gradual onset.

(*b*) By chronic meningitis, with thickening and pressure on the nerves, and sometimes on the cord also. The characteristic symptoms depend upon the damage to both motor and sensory nerves, the former cutting off the muscles and peripheral nerves from the influence of the motor nerve-cells, and hence causing muscular atrophy, very similar to that due to disease of the grey matter, but differing by its association with scattered areas of diminished sensitiveness of the skin. The interference with the reflex loops abolishes reflex action in the part; but if the damage is confined to the upper part of the cord, and the cord itself is pressed upon, there may be an excess of the reflex action on the lower part.

(*c*) Syphilitic disease of vessels may *probably* lead to acute softening, similar to that in the brain. Syphilitic subjects may become suddenly paraplegic, and it is probable that it is by this mechanism, although the fact does not at present rest on post-mortem evidence.

All the above lesions originate in the adventitial (adneural) structures; they are primarily "adneural" diseases. There is, however, a considerable mass of evidence to show that (*d*) diseases which originate in the nerve elements and neuroglia, primarily "neural" diseases, more or less degenerative in character, may be a late effect of syphilis. I believe that in the majority of cases of locomotor ataxy, *i.e.*, of primary posterior sclerosis, there is a history of syphilis.* Anterior cornual degeneration (progressive muscular atrophy) sometimes occurs after constitutional syphilis, and so also do the symptoms associated with sclerosis of the lateral columns. Moreover, I have seen disseminated sclerosis (demonstrated post-mortem) follow constitutional syphilis in a manner which afforded the strongest evidence of a causal relationship. In these cases of degenerative neural disease it does not appear, as far as we can tell, that the anatomical process presents any recognizable difference from that which occurs as a result of other causes; and it is possible that the relation to syphilis, although effective, may not be direct.

Although not strictly a fact of etiology, I may remind you that the effect of treatment often affords an important corroboration of the diagnosis of syphilitic disease. If symptoms, which we have reason to suppose are due to syphilitic disease, improve rapidly when iodide of potassium or mercury is given, the diagnosis is strongly corroborated.† But the converse of this is not equally true. A disease may be due to syphilis, and no improvement be obtained from specific treatment. It must be remembered that as regards (*a*), (*b*), and (*c*), the syphilitic "adneural" disease causes neural changes, softening, degeneration, etc., which are not in any way syphilitic, but are such as would result from any other adneural disease. Under some conditions (of intensity, duration, etc.), the recovery of the nerve-tissue may be impossible, even though the syphilitic adneural disease be completely removed. Further, the diseases of the class (*d*) are not, except in the earliest stage, benefited to any marked extent by anti-syphilitic treatment.

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* See paper on "Syphilitic Neuroses," "Brit. Med. Journal," March, 1879. In this paper I expressed the opinion that in one half of the cases of degenerative locomotor ataxy (excluding cases of gummata in the posterior columns) there is a history of antecedent syphilis. The same opinion has since been expressed by Erb. I now think that this is considerably below the actual proportion. In fourteen cases of ataxy which have come under my notice since the paper was written, eleven had had chancres, and many had presented symptoms of constitutional syphilis.

† Always provided the symptoms are not such as tend to lessen spontaneously. I do not mention this exception in the text, because, important as it is, it has less application to the syphilitic diseases of the spinal cord than to those of the brain.

The exciting causes of disease of the spinal cord sometimes afford diagnostic indications. Exposure to cold may cause acute symptoms, commonly due to inflammatory softening—sometimes focal, sometimes diffuse; and in the latter case accompanied by symptoms of meningitis. It may also cause hæmorrhage. It is especially effective in women at the menstrual period. Repeated exposure may lead to degeneration, especially in the grey matter.

Acute specific diseases, as typhoid fever, are occasionally followed by spinal symptoms, due to changes which are probably of the nature of subacute inflammation. It is very common for a patient after typhoid fever, for a long time, sometimes permanently, to suffer from weakness of the legs; and occasionally during the course of the disease acute symptoms, as those of anterior cornual myelitis, may occur.

Sexual excess is a more common cause of transient functional weakness than of organic disease.

Traumatic influences are frequent causes of cord disease. The cord may be directly pressed upon and damaged by displacement or fracture of the vertebræ, or a severe concussion may be followed by slow paralysis at an interval of a few days or weeks. In such a case I have found, post-mortem, numerous minute foci of chronic inflammation, most abundant in the grey matter. Sometimes a still longer interval elapses between the injury and the paralysis. In such cases a growth or patch of sclerosis appears to be set up by the injury, although years may pass before the symptoms reach a considerable degree of intensity.*

These, then, are the chief etiological facts, which, taken in conjunction with mode of onset and distribution, enable us to form an opinion regarding the nature of the lesion.

To sum up: In examining a case of disease of the spinal cord, the method should be briefly as follows:—First endeavour to ascertain the exact seat of the lesion; note how far the several conducting functions of the cord are impaired, and the highest level of their impairment; then ascertain the condition of the central functions, especially muscular nutrition and irritability, and reflex action, first in the part below the level at which conduction is impaired, and secondly at the supposed level of the lesion: and in this way you may infer, without much difficulty, what is the extent of the lesion transversely and vertically. In the next place endeavour to ascertain its nature by considering—first, how the symptoms came on and developed; secondly, which of the lesions having this mode of onset and development, occur in the region affected; and thirdly, which of them are produced by the cause or causes to which the disease is apparently due.

This process of diagnosis may seem somewhat elaborate, and, no doubt, a practised observer does not always consciously go through it. But, in most cases, if he wish to avoid error, he goes through it unconsciously, and no step can be with safety dispensed with. We may thus, in almost all cases, arrive at an exact diagnosis of the seat of the disease, and, in a large number of cases, of its nature also. There are, however, some cases with respect to which the diagnosis of the nature of the lesion can be approximate only, although we can always limit it to one or two possibilities.

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* An instructive instance of the way in which the results of an injury of the head may cause both growths and arterial disease, and, years later, symptoms both chronic and acute, will be found recorded in the author's "Medical Ophthalmoscopy," Case 4, p. 248.

Functional Diseases.—We may now understand the principles of the diagnosis of the "functional" diseases of the cord. They are of two classes. The symptoms in one class are confined to functions which have to do with will—consciousness; there is loss of voluntary power or loss of sensation, but no objective indication of interference with the functions of the cord; nutrition and reflex action are normal. These are the cases of "hysterical," "emotional," "ideal" paralysis, and they are rather functional diseases of that part of the brain which has to do with the movement or sensations of the limbs, than functional diseases of the cord. They are distinguished (1) by the absence of all signs of alteration in the functions of the cord as a central organ; (2) by the manner in which they come on, often sudden, or succeeding transient symptoms of the same kind; (3) by their occurrence in individuals who present other symptoms of "hysteria," or are of an age and sex in which such symptoms are common (young women, girls, or boys); (4) by their increase when attention is directed to them, and decrease when they are disregarded, or when treatment is employed which produces a strong impression on the psychical centres.

For instance, a member of a religious sisterhood, after having been kept in bed for some time on account of a uterine displacement, suddenly found that she could not stand. Every sign of derangement of the functions of the cord as a central organ was absent. After considerable search for loss of sensibility, a patch of anæsthesia was found on the right thigh. The next day there was a great increase in the anæsthetic area, without any increase in the motor symptoms. I advised that she should be encouraged to get up, and that little notice should be taken of her weakness, and that her sensibility should not be again tested. In a week she was practically well. Doubtless, by repeated examination, she might easily have been kept anæsthetic for months, and then have been cured by magnets or metals. Again, a little girl, aged eleven, suddenly became paraplegic, and next day was carried into the hospital consulting-room by a sympathetic sister. All symptoms of impairment of the central function of the cord or of sensation were absent. She was known to be "hysterical," for she had been under treatment for severe hystero-epileptic fits. Her legs were well faradized, and she walked from the hospital home, a distance of three miles, without difficulty.

But, as I said before—and the importance of the point must be my excuse for saying it again—we must be sure that there is no evidence of organic disease before we regard the affection as purely hysterical. Not only do symptoms of hysteria occur in the subjects of organic disease, but it is not uncommon for slight organic disease to underlie an hysterical affection and determine its direction, just as in hypochondriasis, slight real disease in some organ often determines other symptoms which are in a sense unreal.

The second class of functional diseases consist in transitory impairment of the functions of the cord itself, and sometimes of the central functions. The distinction of such from organic disease may be most difficult, and probably, indeed, has not any real basis in pathology. The changes which in one patient are transient, may in another be permanent. We have to rely for our diagnosis on the slight degree and variable character of the symptoms, and on the presence of some condition known to be a cause of such symptoms. The influences to which the cases I have seen have been due have been especially alcohol, and gout, and sexual excesses. Very often there is an entire absence of the signs of impairment of the central functions, and then we can be more confident. Often, however, in these cases, we must be content to wait a time before we express a positive opinion.

It will be observed that I have said nothing of "anæmia of the cord," of "hyperæmia of the cord," or of "reflex paralysis." In current descriptions of the symptoms of these conditions, I cannot help thinking that a vigorous scientific imagination has contributed much more than observation has supplied. The only practical knowledge of the effects of anæmia and hyperæmia of the cord, is, that they seem capable of causing such disturbance of the sensory structures as reveals itself in subjective sensations of tingling, pins and needles, and the like, and perhaps also some impairment of motor conduction. A large number of authorities here and abroad are sceptical as to the existence of such a condition as "reflex paralysis," *i.e.*, a paralysis due to the effect on the centre of some peripheral irritation, disappearing when this was removed. I have never seen a case which seemed to me distinctly such; and, although our modern knowledge of the various phenomena of inhibition and reflex action renders such a paralysis *à priori* even probable, it is certain that the theory has been extensively misapplied.

Spinal Meningitis.—The object of this lecture has been to explain the principles of the diagnosis of diseases of the cord itself. But it may be well to allude briefly to the diagnosis of spinal meningitis. Of acute meningitis I need say little. The acute symptoms, spinal pains and severe spasms, are well known. Chronic spinal meningitis, however, is a disease regarding which current opinion has curiously changed during the last fifteen years. A large number of symptoms were assigned to chronic meningitis which we now know have nothing to do with that pathological state. I have mentioned that to it the symptoms of chronic spasm, "spasmodic paraplegia," were ascribed. But we now know that these are due to alterations within the cord, and are independent of any meningitis. The only symptoms which are usually due to this condition are those which result from the involvement of the nerve-roots in their passage through the diseased membranes. The roots are irritated by the adjacent inflammation. The meninges often become much thickened, and the change is then called "pachy-meningitis." In this thickening, the nerve-roots are often greatly damaged by pressure. The irritation affects first the sensory roots, causing "excentric" pains and hyperæsthesia, to which are often added areas of anæsthesia here and there, due to the greater damage of some nerve-roots. The affection of the motor roots causes symptoms similar to those of disease of the anterior cornua, but very irregular in distribution. The peripheral motor nerve-fibres, cut off from their motor cells, degenerate, and the muscular fibres waste, and present electrical reactions which vary according to the rapidity of the morbid process. Sometimes the nutrition of the skin suffers. There is often, in addition, pain in the back, from the lumbar to the cervical region, sometimes severe between the shoulders.

The chief conditions with which chronic spinal meningitis may be confounded are posterior sclerosis (locomotor ataxy) in which the sensory nerve-roots are implicated, and anterior cornual degeneration (progressive muscular atrophy). From the former it is distinguished by the absence of ataxy, from the latter by the irregular distribution of the symptoms, and from both by the existence of hyperæsthesia, of limited areas of anæsthesia, and of extensive spinal pain. It must be remembered that inflammation often affects the substance of the cord as well as the meninges, or the cord may be pressed upon by the thickened membranes, and so mixed symptoms may result.

A word on the subject of the nomenclature of diseases of the spinal cord. If we wish to obtain clear ideas, it is essential to use terms, where we can, which shall be pathological, and which shall be at once simple and descriptive. To obtain these, we must avoid the error, too common, of striving after extreme brevity. Terms, the meaning of which is obvious, are, even if somewhat longer, to be preferred to shorter expressions the meaning of which is obscure. We are apt to associate with brief obscure names the idea of definite diseases.

But if we would gain and convey exact ideas of the diseases of the spinal cord, we must endeavour to substitute the idea of morbid processes for that of definite diseases.

A simple and convenient system of terminology lies close to hand. We have in the spinal cord, the two cornua of grey matter and the three columns, lateral, anterior, and posterior. In each of these situations the various morbid processes already described may occur, and we have only to combine the terms indicating the place and the lesion to have a system of terminology already partly in use, and which will altogether suffice for our present needs. Thus we may have a columnal or a cornual myelitis, hæmorrhage, sclerosis, degeneration, or growth. We may have, for instance, an "anterior cornual myelitis," or, for shortness (since we cannot yet diagnose posterior cornual diseases), a "cornual myelitis;" or we may have a cornual degeneration. For anterior cornual myelitis, the term "tephro-myelitis" has been proposed by Charcot, and "anterior polio-myelitis" by Erb. The latter term is obtaining wide currency, but its meaning is much less obvious than that of "anterior cornual myelitis," while it does not possess the advantage of a single syllable. The simpler system of nomenclature I have employed throughout this lecture, and it has probably been readily intelligible, although unexplained.

V.—ILLUSTRATIONS OF DIAGNOSIS.

The following illustrations may render clearer the application of the methods of diagnosis. I will take, first, two cases in which the pathological diagnosis presented no difficulty, since both were cases of fractured spine, and complete paralysis of the legs occurred immediately on the accident, indicating direct damage to the cord by the displaced bone.

(1.) In the one case, that of a sailor, there was no irregularity of the spines to guide us as to the position of the injury, out this was clear enough from the symptoms. The legs were completely paralysed, and all the muscles, when the patient came under observation, some months after the injury, were greatly wasted, faradaic irritability being extinct. This proved complete degeneration of the motor nerves arising from the lumbar enlargement. Sores had formed on the limbs and sacrum, indicating damage to the nerves which influence the nutrition of the skin. Sensation was at first lost, but afterwards returned as hyperæsthesia—suggesting initial damage and partial recovery of the nerves or tracts conveying sensation. The sphincters were powerless, and their condition was such as to indicate damage to, or separation from, their centres in the cord. From these symptoms we inferred damage by compression of the lower part of the lumbar enlargement, and of the nerves passing it. But what was the state of the dorsal region of the cord? Sensation above the groins was normal, but this does not exclude slight damage to the cord, since the impairment of sensation caused by slight damage may soon pass away. Here it was that the superficial trunk reflexes assisted us. We found that the epigastric reflex and the abdominal reflex were perfectly natural on each side, even in the lower part of the abdomen. The cremasteric reflex, however, was active on the right side, absent on the left; so that we had evidence that the dorsal cord was normal, and that the damage commenced at the 1st lumbar nerve, where the reflex loops were damaged on the left side and normal on the right, and that just below this point the damage was great. The patient died, and the autopsy revealed exactly the condition which had been diagnosed. The dorsal cord was uninjured, and so was the highest part of the lumbar enlargement; while its lower portion was split in two by a fracture, with displacement, of the 1st lumbar vertebra, which had also compressed the nerve-roots. Microscopical examination revealed, also, slighter mischief in the cord, extending as far as the upper part of the lumbar enlargement.

(2.) The other case is that of a girl, who is now in

University College Hospital under the care of Mr. Heath. She fell off a house-top and became at once paraplegic. There were indications of damage to the bones about the 10th dorsal vertebra. The legs were completely paralysed; but there was only slight wasting, the faradaic irritability of the muscles being preserved, although lowered; and reflex action was preserved. Hence it was inferred that the motor nerves were undegenerated, that the lumbar anterior cornua were not directly damaged, that the reflex loops were entire; in short, that the damage to the cord was at or above the highest part of the lumbar enlargement. There was loss of sensibility to pain in the legs, that to touch being perfect. Hence we inferred that the destruction of the cord was incomplete. This loss extended as high as the epigastrium—evidence of some damage to the cord as high as the origin of the 8th dorsal nerves. This was corroborated by the condition of the superficial reflexes of the trunk: the abdominal was lost on both sides; the epigastric was lost on the right side, but present on the left, indicating clearly the highest level of damage. Thus there was evidence of affection of the cord from the origin of the 8th to that of the 11th dorsal pairs; but the symptoms did not show whether the damage was equal throughout this region. This information was, however; supplied by an examination of the faradaic irritability of the abdominal muscles. Above the umbilicus there was normal irritability; below the umbilicus it was gone—*i.e.*, the motor fibres of the 9th pair were undegenerated, their anterior cornua were undamaged, the fibres of the 10th pair, perhaps also the 11th pair, were degenerated, and the corresponding cornua probably damaged. As the lumbar enlargement was not directly damaged, we were able thus to limit with precision the considerable damage to the cord to the origin of the 10th, or 10th and 11th pair. The loss of the epigastric reflex on the right side indicated that the damage to the cord on that side was greater than on the left. The subsequent progress of the case showed the significance of these indications. A month later the epigastric reflex returned on the right side, an indication of commencing recovery in the upper part of the damaged region. A few weeks later she gained some power of moving the left leg, but the reflex action in the legs, deep and superficial, became excessive. Now, four months after the injury, the abdominal reflexes are returning; a slight reflex can be obtained just above the groin, and above the umbilicus, none at or just below the umbilicus.

In both these cases, thus, the information conveyed by the trunk reflexes was most important.

(3.) A man, aged twenty-eight, had suffered from weakness of the legs for more than two years. He was able to walk leaning forwards upon his sticks. His arms were unaffected. He could just flex the hips and extend the knees, but could not flex the knees, and scarcely the ankles. The right leg was the weaker. The legs were well nourished. Even as he entered the room, the clonic spasm at the ankle-joint, as the calf-muscles were put on the stretch, was conspicuous, and it was found that the knee reflex was in great excess, and the ankle clonus could be obtained by the slightest pressure against the soles. A slight peripheral impression caused rigid spasm, succeeded by clonic contraction as the stronger spasm passed lessened (spinal epilepsy, see p. 44). Thus the loss of power showed interruption to the motor path somewhere below the cervical enlargement. The preservation of the deep reflexes, and the absence of wasting in the legs, showed the integrity of the lumbar reflex loops and grey matter, while the intensification of these reflexes pointed to such overaction of the reflex centres as accompanies descending degeneration in the lateral columns.

The next point was to search for any evidence of mischief beyond the motor tract. This was found in the fact that sensation to pain in the legs was perverted: the pain of a prick was felt, but in an abnormal manner. Sensation to touch was normal. Thus it was evident that somewhere the sensory tract also was interfered with to a slight degree. The same fact was indicated by a sense of constriction around the abdomen. These two symptoms pointed to a lesion extending beyond the motor tracts, *i.e.*, to a focal lesion, and the fact that the sense of constriction was around the lower part of the abdomen made it probable that the lesion was in the lower part of the dorsal region. The superficial reflexes of the trunk were then examined. On the left side the epigastric reflex and the abdominal reflex above the umbilicus were very active. Just below the level of the umbilicus the abdominal reflex was much lessened, and midway between the umbilicus and groin could not be obtained. On approaching Poupart's ligament it was again produced, and an impression here caused reflex flexion of the hip. Behind, the left dorsal reflex was active, the lumbar absent. On the right side, however, the abdominal reflex was extremely slight throughout, and the epigastric reflex could not be produced (although so active on the left side), and no lower dorsal or lumbar reflex was obtainable. Thus the reflex phenomena pointed to a very limited lesion on the left side, at the level of origin of the 11th dorsal nerve, while on the right side the more extensive loss indicated more extensive mischief in the right half of the lower dorsal cord, corresponding to the greater weakness of the right leg. This affection of the reflex in the left side corresponded to the position of the constricting band around the lower part of the abdomen. What was the nature of the lesion? Its onset was very gradual; the commencing was by a sensation of "numbness," followed, eight months later, by weakness. This extreme slowness pointed to degenerative changes—a local patch of "sclerosis." There was no bone disease, no history of syphilis; but the patient had been much exposed to wet two months before the onset, and we have seen that degenerative changes sometimes result from this cause.

(4.) A man, aged forty-eight, came under treatment for weakness of the right leg, chiefly marked in the movements of the foot. The corresponding arm was unaffected. Thus there was impairment of the motor conducting tract (lateral column) on the right side, somewhere below the cervical enlargement. Sensation was unimpaired; the sensory tract, therefore, undamaged. The nutrition of the legs was good; the plantar reflex ready and equal; the knee reflex excessive in each leg; the ankle clonus and front-tap contraction obtainable in each. Hence the reflex loops and grey matter of the chief part of the lumbar enlargement were intact; the excess showed that there was probably lateral sclerosis descending from a lesion above, and that this existed on the left side as well as on the right. The lesion was thus evidently somewhere in the dorsal region of the cord. To learn its seat further, the higher superficial reflexes were examined. The cremaster reflex was distinct on the left side, not to be obtained on the right. The abdominal reflexes were normal on the left side, but on the right they could not be obtained except just below the edge of the ribs. The epigastric reflex, excited from the side of the chest, was distinct on each side—as readily excited on the right side as on the left. Hence we had evidence, from the impairment of the reflex loops, that the cord was damaged on the right side, from the 8th dorsal nerve to the 1st lumbar. The excess of the deep reflexes in the left leg suggests that the mischief had implicated slightly the left half of the cord, so as to lead to some descending degeneration, although not to any loss of the superficial reflexes. It is possible, however, that the descending degeneration in the left side of the lumbar enlargement, may have been due to the damage of fibres of the direct pyramidal tract on the right side, before their decussation to the left side.

So much for the anatomical diagnosis. The affection had come on acutely. In nine hours from the first symptom the leg was powerless, and it was five months before it recovered. The "acute" onset points to either a vascular lesion or inflammation. Causal indications were obscure. The man attributed his symptoms to a severe strain three days before, which he stated also caused "ulcerated bowels."

The wide extent of the lesion in the right side of the cord suggests the probability of a myelitis, not severe in degree, although acute in onset, rather than of hæmorrhage.

(5.) A young man, aged twenty-two, presented himself with weakness, wasting, and deformity of the left forearm. The muscles of the shoulder were normal. The upper arm muscles were rather smaller than those of the right side, but were of fair size and nutrition. The muscles of the forearm were greatly wasted, except the radial extensor of the wrist; the muscles of the thumb and little finger were much wasted, the interossei only slightly. The wasted muscles had lost faradaic and voltaic irritability, the condition being of long-standing, the muscular fibres were probably totally degenerated. Sensation was perfect. The leg was not quite so strong as the other; nutrition normal; the deep reflexes excessive in each leg; knee reflex increased, and ankle clonus and front-tap contraction obtainable. Thus we had evidence, from the muscular wasting, of a limited lesion in the left anterior cornu in the lower part of the cervical enlargement; from the impaired power in the leg, of slight damage to the motor tract for the leg; and from the excess of the deep reflexes, of degeneration in the pyramidal tracts descending from above.

What was the nature of the lesion? Its onset was sudden, nine years before. One morning he suddenly felt a pain in the back of his neck, then he found his arms and legs becoming weak, and this increased so rapidly, that in half an hour or so he was unable to move any limb, but there was no noticeable loss of sensation. Thus he lay for three weeks, and then the right arm began to regain power, next the right leg, and then the left leg, so that in two months he could walk. The wasting in the muscles of the left arm was very rapid. There was no causal indication. Thus there was a lesion of sudden onset, and therefore primarily vascular—thrombosis or hæmorrhage—at first affecting a wide transverse area of the cord, and impairing all its functions at the spot, except conduction of sensation. The part slightly damaged soon recovered, but there remained an area of considerable damage in the left anterior cornu, and of slighter damage in the adjacent conducting tract to the leg. The focus of disease was no doubt the seat of the primary lesion; probably a local extravasation interfering with the opposite side of the cord by pressure.

(6.) The following case is somewhat complex in its indications, but is instructive as affording an illustration of the diagnosis of chronic pachy-meningitis, *i.e.*, chronic meningitis with thickening. For an opportunity of seeing the patient, I am indebted to Dr. Russell, of Birmingham.

A man, aged forty-seven, complained of weakness of the legs, which was found, on examination, to be of irregular distribution. In both legs the muscles moving the hip-joint possessed good power. The flexors and extensors of the knee-joint were strong in the right, but very weak in the left leg. The flexors of the ankle in the left leg were rather weak, in the right were powerless; the extensors of the ankle were weak in both legs, but much weaker in the right than the left. The muscles were wasted, and had lost faradaic irritability in proportion to their weakness, the voltaic irritability being preserved. The wasting was greatest in the extensors of the left knee, and flexors of the right ankle. In the latter, faradaic irritability was gone. Sensation was normal, except in an area in the front of the left leg, where it was absolutely lost to both touch and pain. Reflex action; plantar normal; no ankle clonus; knee reflex slight in the right leg, absent in the left.

The affection of nutrition and electrical irritability indicated disease in the anterior cornua, or in the motor nerves springing from them. By this the impairment of the knee reflex was also explained. The weakness was in proportion to the wasting; hence there was no reason to infer other disease than that interfering with nutrition. The diagnosis thus lay between a primary cornual disease, and damage to the nerve-roots by meningeal changes. The patch of anæsthesia in the left leg was in favour of the latter. It is rare that anterior cornual disease impairs sensation.

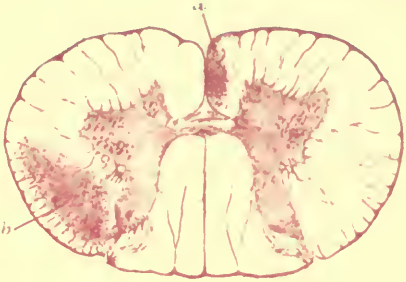
The mode of onset was then investigated to ascertain what light the order of the symptoms would throw on the seat, and their rapidity of development on the nature of the disease. The first symptoms commenced nine months before, and were sensory; soreness in the left leg, followed by shooting pains, sometimes in the big toe, sometimes in the calf, but confined to the left leg; these lasted for two months, and during that time the leg gradually got weak. After this similar pains were felt in the right leg, and this also became weak.

Pains of this darting character are usually due to irritation of the sensory nerve-roots; they are similar to those met with in locomotor ataxy, and are almost unknown in affections of the anterior cornua. These pains, in conjunction with the patch of anæsthesia, rendered it highly probable that the mischief was outside the cord in the meninges—chronic meningitis with thickening, the nerve-roots being irregularly damaged by irritation and compression. Hence a careful search was made to ascertain if there were any trunk symptoms, throwing light on the disease. Inquiry elicited a history of shooting pains on the right side of the trunk, at the level of the epigastrium, with a unilateral sense of constriction. Some local hyperæsthesia was found, but no anæsthesia. This also pointed to local irritation of the posterior nerve-roots, higher up —to irregular meningeal irritation.

The causal influences were then ascertained. No immediate cause could be ascribed except general bad health. Chronic pachy-meningitis is, in the immense majority of cases, due to bone-disease or syphilis. There was no evidence of bone-disease, but the patient had had a chancre twenty years before.

Thus the motor symptoms indicated either cornual or meningeal disease; the sensory symptoms pointed strongly to the latter, and the causal conditions, as far as they went, harmonized with the view. (The patient had had some sugar in the urine, which, except for rendering the prognosis worse, had little apparent bearing on the case.) He had taken iodide for a time, but without improvement. This did not, however, militate against the diagnosis, for the following reason. The meningeal change, though probably syphilitic, had caused damage to the nerves, which, descending as degeneration, had led to secondary changes in the muscles. The removal of the syphilitic change in the membranes would not at once restore the nerves. Their regeneration, if still possible, would be, of necessity, a work of time, perhaps of more time than had yet been allowed. Hence the patient was urged to persevere with the iodide, and mercury was added to it, and he was advised to continue the use of a voltaic battery to the muscles. A month later there was slight but distinct increase of power in the left thigh. I did not see him again, but a year later he was seen, incidentally, by Dr. Russell, who has been good enough to inform me that the man then said he was, and appeared to be, perfectly well. The result thus affords a strong confirmation of the diagnosis.

Gentlemen,—to know our enemy is, if not "half the battle," at least a most important part of it. To understand the disease with which we have to deal is only second in importance to the knowledge of how to treat it. Real advance in therapeutic science comes but slowly. The present generation has, however, witnessed not unimportant progress, and still greater additions in our knowledge of the nature of disease, and in our means of precise diagnosis. I shall be glad if the account I have endeavoured to give of one department of diagnosis may help any of you in your daily work, by rendering to you the problem of the diagnosis of diseases of the spinal cord, if not more simple, less obscure.



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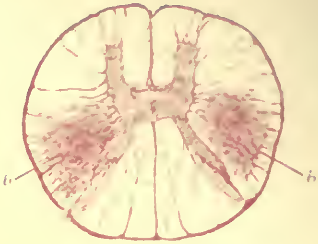

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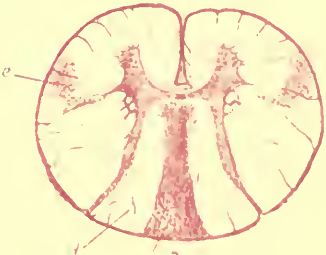

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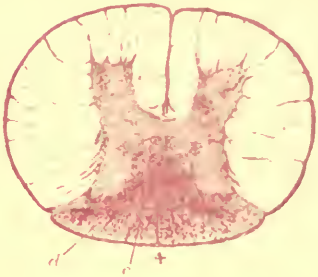

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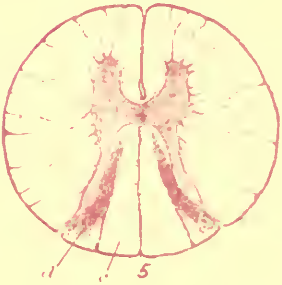

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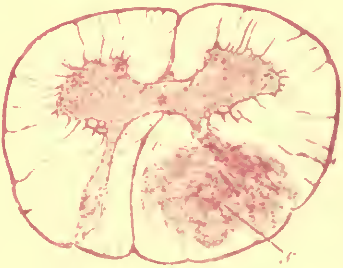

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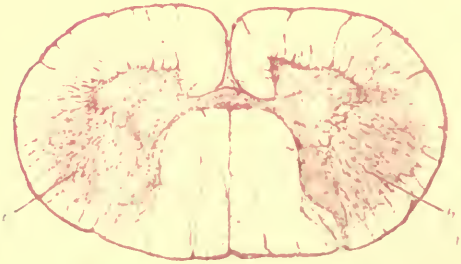

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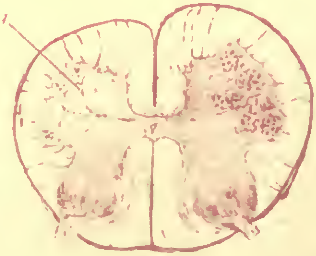

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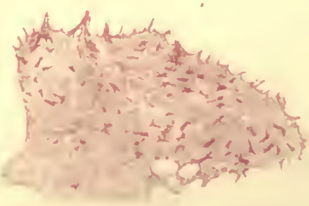

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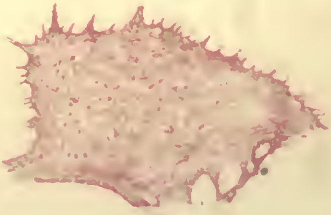

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DESCRIPTION OF PLATE.

THE figures represent some of the more important lesions of the spinal cord. Although semi-diagrammatic, they have, with one exception, been drawn, with care, from actual sections. The exception is Fig. 2, which is after Charcot. It may be well to state, for the information of those unfamiliar with the process of microscopical examination of the nerve centres, that when a section of the spinal cord is stained with carmine, the tint assumed by the different parts varies, and conveys important information. The grey substance stains much more deeply than the white, and the nerve-cells more deeply than the inter-cellular grey substance. Hence the grey matter appears like an H-shaped rose-coloured area, in which the nerve-cells appear of a much deeper red. The white substance of the nerve-fibres does not stain, and although the axis cylinders stain, they are not sufficient in bulk to give much colour to the white columns. Connective tissue, however, stains very deeply, and the edge of the section (pia mater) is thus deep red, and so are the trabeculæ of connective tissue which extend into the white substance. Since the process of sclerosis consists in an atrophy of the nerve-fibres and an increase in the connective tissue, areas so affected stain deeply in proportion to the intensity of the change, and its existence and degree may thus be rendered conspicuous even to the naked eye. The relative tint of the figures is nearly that of the sections from which they were drawn, all of which were stained with carmine. The letters indicating corresponding parts are the same in all the figures.

FIG. 1. *Descending Degeneration, unilateral.*—Section of spinal cord, cervical region, from a case of left hemiplegia due to disease of the right cerebral hemisphere. The two pyramidal tracts are degenerated, viz., the small "direct pyramidal tract" (*a*), close to the anterior median fissure, on the right side of the cord; and the "crossed pyramidal tract" (*b*) in the opposite lateral column. This degenerated tract is seen not to extend up to the surface, being bounded by the so-called "cerebellar tract." (See p. 11.)

FIG. 2. *Descending Degeneration, bilateral.*—Section of spinal cord, dorsal region, below a point damaged by compression. Both lateral columns crossed pyramidal tracts (*b b*) are degenerated. There is no degeneration in the direct tracts, which had probably ceased (by decussation) above the level of the section, or may have been absent.

FIG. 3. *Ascending Degeneration.*—Section of spinal cord in dorsal

region, from a case in which the lower extremity of the cord was crushed by a fracture of the spine. The postero-median columns (*c*) are densely sclerosed. The postero-external columns (*d*) are quite free from sclerosis. The pyramidal tracts in the lateral columns are seen to be also free from disease (compare Figs. 2 and 3), but just in front of each is a symmetrical area of slight degeneration (*e*). (See p. 13.)

FIG. 4. *Posterior Sclerosis, Locomotor Ataxy.*—Section at the level of the first lumbar nerves. The posterior columns are densely sclerosed throughout their entire extent. The remaining white columns and anterior cornua are healthy.

FIG. 5. *Sclerosis of postero-external column* (*posterior root-zone*), *Locomotor Ataxy* (from a section prepared by Prof. Pierret, of Lyons).—A dense band of sclerosis occupies the postero-external column (*d*), through which the posterior nerve-roots pass. The postero-median columns (*c*) are free from sclerosis. The bands of sclerosis are narrow, probably from the contraction of the tissue, since, from the position of the limiting septum, they appear to occupy the entire width of this column. The patient suffered from well-marked locomotor ataxy.

FIG. 6. *Syphilitic growth in posterior column.*—Section through the spinal cord, cervical region, of a man who died from syphilitic disease of the brain. A growth (*f*) occupies the right postero-external column, and has enlarged it to three times the normal size, displacing the posterior median septum to the left. The growth has invaded the right posterior cornu, and extended a little way beyond it into the lateral column. It caused inco-ordination, and partial loss of sensibility in the right arm.

FIG. 7. *Anterior cornual degeneration.*—Section of spinal cord, cervical region, from a patient suffering from progressive muscular atrophy. The grey substance of the anterior cornua is degenerated and irregularly translucent, the nerve-cells having disappeared (compare also Figs. 9 and 10). The lateral columns (pyramidal tracts) are also sclerosed. The posterior columns are healthy.

FIG. 8. *Anterior cornual myelitis* (*Infantile Paralysis*).—Section of spinal cord, lumbar enlargement, from a case of old infantile paralysis of the left leg. The whole left half of the cord is smaller than the right. The left anterior cornu is shrunken, and presents evidence of previous inflammation. The tissue is degenerated and translucent, containing large vessels. All the motor nerve-cells, so conspicuous on the other side, have disappeared.

FIG. 9. Normal anterior cornu, cervical region, showing numerous multipolar nerve-cells.

FIG. 10. Anterior cornu, same position, from a case of progressive muscular atrophy. All the nerve-cells have disappeared; minute shrunken corpuscles here and there are probably their remains. The grey matrix, instead of being uniform, is irregular, translucent at some spots, unduly dense, from sclerosis, at others, especially near the edge of the cornu.

London, New Burlington Street.
April, 1880.

SELECTION

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